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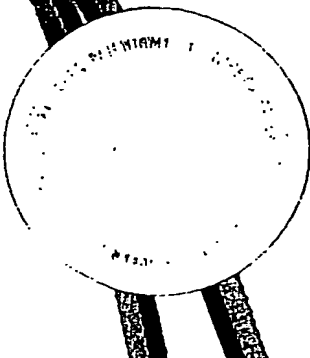
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Methods and compositions for the response prediction of malignant neoplasia to treatment

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METHODS AND COMPOSITIONS FOR THE RESPONSE PREDICTION OF MALIGNANT NEOPLASIA TO TREATMENT

TECHNICAL FIELD OF THE INVENTION

5 The invention relates to methods and compositions for the prediction, diagnosis, prognosis, prevention and treatment of neoplastic disease. Of particular interest is the response prediction of neoplastic lesions to various therapeutic regimens. Neoplastic disease is often caused by chromosomal rearrangements which lead to over- or underexpression of the rearranged genes. The invention discloses genes which are overexpressed in neoplastic tissue and are useful as diagnostic markers and targets for treatment. Methods are disclosed for predicting, diagnosing and prognosing as well as preventing and treating neoplastic disease.

BACKGROUND OF THE INVENTION

Chromosomal aberrations (amplifications, deletions, inversions, insertions, translocations and/or viral integrations) are of importance for the development of cancer and neoplastic lesions, as they account for deregulations of the respective regions. Amplifications of genomic regions have been described, in which genes of importance for growth characteristics, differentiation, invasiveness or resistance to therapeutic intervention are located. One of those regions with chromosomal aberrations is the region carrying the HER-2/neu gene which is amplified in breast cancer patients. In approximately 25% of breast cancer patients the HER-2/neu gene is overexpressed due to gene amplification. HER-2/neu overexpression correlates with a poor prognosis (relapse, overall survival, sensitivity to therapeutics). The importance of HER-2/neu for the prognosis of the disease progression has been described [Gusterson et al., 1992, (1)]. Gene specific antibodies raised against HER-2/neu (Herceptin™) have been generated to treat the respective cancer patients. However, only about 50% of the patients benefit from the antibody treatment with Herceptin™, which is most often combined with chemotherapeutic regimen. The discrepancy of HER-2/neu positive tumors (overexpressing HER-2/neu to similar extent) with regard to responsiveness to therapeutic intervention suggest, that there might be additional factors or genes being involved in growth and apoptotic characteristics of the respective tumor tissues. There seems to be no monocausal relationship between overexpression of the growth factor receptor HER-2/neu and therapy outcome. In line with this the measurement of commonly used tumor markers such as estrogen receptor, progesterone receptor, p53 and Ki-67 do provide only very limited information on clinical outcome of specific therapeutic decisions. Therefore there is a great need for a more detailed diagnostic and prognostic classification of tumors to enable improved therapy decisions and prediction of survival of the patients. The present invention addresses the

need for additional markers by providing genes, which expression is deregulated in tumors and correlates with clinical outcome. One focus is the deregulation of genes present in specific chromosomal regions and their interaction in disease development and drug responsiveness.

5 HER-2/neu and other markers for neoplastic disease are commonly assayed with diagnostic methods such as immunohistochemistry (IHC) (e.g. HercepTest™ from DAKO Inc.) and Fluorescence-In-Situ-Hybridization (FISH) (e.g. quantitative measurement of the HER-2/neu and Topoisomerase II alpha with a fluorescence-*in-situ*-Hybridization kit from VYSIS). Additionally HER-2/neu can be assayed by detecting HER-2/neu fragments in serum with an ELISA test (BAYER Corp.) or a with a quantitative PCR kit which compares the amount of HER-2/neu gene
10 with the amount of a non-amplified control gene in order to detect HER-2/neu gene amplifications (ROCHE). These methods, however, exhibit multiple disadvantages with regard to sensitivity, specificity, technical and personnel efforts, costs, time consumption, inter-lab reproducibility. These methods are also restricted with regard to measurement of multiple parameters within one patient sample ("multiplexing"). Usually only about 3 to 4 parameters (e.g. genes or gene
15 products) can be detected per tissue slide. Therefore, there is a need to develop a fast and simple test to measure simultaneously multiple parameters in one sample. The present invention addresses the need for a fast and simple high-resolution method, that is able to detect multiple diagnostic and prognostic markers simultaneously.

SUMMARY OF THE INVENTION

20 The present invention is based on discovery that chromosomal alterations in cancer tissues can lead to changes in the expression of genes that are encoded by the altered chromosomal regions. Exemplary 43 human genes have been identified that are co-amplified in neoplastic lesions from breast cancer tissue resulting in altered expression of several of these genes (Tables 1 to 4). These 43 genes are differentially expressed in breast cancer states, relative to their expression in normal,
25 or non-breast cancer states. The present invention relates to derivatives, fragments, analogues and homologues of these genes and uses or methods of using of the same.

The present invention further relates to novel preventive, predictive, diagnostic, prognostic and therapeutic compositions and uses for malignant neoplasia and breast cancer in particular. Especially membrane bound marker gene products containing extracellular domains can be a
30 particularly useful target for treatment methods as well as diagnostic and clinical monitoring methods.

It is a discovery of the present invention that several of these genes are characterized in that gene products functionally interact in signaling cascades or by directly or indirectly influencing each other. This interaction is important for the normal physiology of certain non-neoplastic tissues (e.g. brain or neurogenic tissue). The deregulation of these genes in neoplastic lesions where they are normally exhibit of different level of activity or are not active, however, results in pathophysiology and affects the characteristics of the disease-associated tissue.

The present invention further relates to methods for detecting these deregulations in malignant neoplasia on DNA and mRNA level.

The present invention further relates to a method for the detection of chromosomal alterations characterized in that the relative abundance of individual mRNAs, encoded by genes, located in altered chromosomal regions is detected.

The present invention further relates to a method for the detection of the flanking breakpoint named chromosomal alterations by measurement of DNA copy number by quantitative PCR, DNA-Arrays and DNA sequencing.

A method for the prediction, diagnosis or prognosis of malignant neoplasia by the detection of DNA sequences flanking named genomic breakpoint or are located within such.

The present invention further relates to a method for the detection of chromosomal alterations characterized in that the copy number of one or more genomic nucleic acid sequences located within an altered chromosomal region(s) is detected by quantitative PCR techniques (TaqMan™, Lightcycler™ and iCycler™).

The present invention further relates to a method for the prediction, diagnosis or prognosis of malignant neoplasia by the detection of at least 2 markers whereby the markers are genes or fragments thereof or genomic nucleic acid sequences that are located on one chromosomal region which is altered in malignant neoplasia and breast cancer in particular.

The present invention also discloses a method for the prediction, diagnosis or prognosis of malignant neoplasia by the detection of at least 2 markers whereby the markers are located on one or more chromosomal region(s) which is/are altered in malignant neoplasia; and the markers interact as (i) receptor and ligand or (ii) members of the same signal transduction pathway; (iii) members of synergistic signal transduction pathways or (iv) members of antagonistic signal transduction pathways or (v) transcription factor and transcription factor binding site.

Also disclosed is a method for the prediction, diagnosis or prognosis of malignant neoplasia by the detection of at least one marker whereby the marker is a VNTR, SNP, RFLP or STS which is located on one chromosomal region which is altered in malignant neoplasia due to amplification and the marker is detected in (a) a cancerous and (b) a non cancerous tissue or biological sample from the same individual. A preferred embodiment is the detection of at least one VNTR marker of Table 6 or at least on SNP marker of Table 4 or combinations thereof.. Even more preferred can the detection, quantification and sizing of such polymorphic markers be achieved by methods of (a) for the comparative measurement of amount and size by PCR amplification and subsequent capillary electrophoresis, (b) for sequence determination and allelic discrimination by gel electrophoresis (e.g. SSCP, DGGE), real time kinetic PCR, direct DNA sequencing, pyro-sequencing, mass-specific allelic discrimination or resequencing by DNA array technologies, (c) for the dertermination of specific restriction patterns and subsequent electrophoretic separation and (d) for allelic discrimination by allele specific PCR (e.g. ASO). An even more favorable detection of a hetrozygous VNTR, SNP, RFLP or STS is done in a multiplex fashion, utilizing a variety of labeled primers (e.g. fluorescent, radioactive, bioactive) and a suitable capillary electrophoresis (CE) detection system.

In another embodiment the expression of these genes can be detected with DNA-arrays as described in WO9727317 and US6379895.

In a further embodiment the expression of these genes can be detected with bead based direct flourescent readout techniques such as described in WO9714028 and WO9952708.

In one embodiment, the invention pertains to a method of determining the phenotype of a cell or tissue, comprising detecting the differential expression, relative to a normal or untreated cell, of at least one polynucleotide comprising SEQ ID NO: 2 to 6, 8, 9, 11 to 16, 18, 19 or 21 to 26 or 53 to 75, wherein the polynucleotide is differentially expressed by at least about 1.5 fold, at least about 2 fold or at least about 3 fold.

In a further aspect the invention pertains to a method of determining the phenotype of a cell or tissue, comprising detecting the differential expression, relative to a normal or untreated cell, of at least one polynucleotide which hybridizes under stringent conditions to one of the polynucleotides of SEQ ID NO: 2 to 6, 8, 9, 11 to 16, 18, 19 or 21 to 26 or 53 to 75 and encodes a polypeptide exhibiting the same biological function as given in Table 2 or 3 for the respective polynucleotide, wherein the polynucleotide is differentially expressed by at least at least about 1.5 fold , at least about 2 fold or at least about 3 fold.

5 In another embodiment of the invention a polynucleotide comprising a polynucleotide selected from SEQ ID NO: 2 to 6, 8, 9, 11 to 16, 18, 19 or 21 to 26 and 53 to 75 or encoding one of the polypeptides with SEQ ID NO: 28 to 32, 34, 35, 37 to 42, 44, 45 or 47 to 52 or 76 to 98 can be used to identify cells or tissue in individuals which exhibit a phenotype predisposed to breast cancer or a diseased phenotype, thereby (a) predicting whether an individual is at risk for the development, or (b) diagnosing whether an individual is having, or (c) prognosing the progression or the outcome of the treatment malignant neoplasia and breast cancer in particular.

10 In yet another embodiment the invention provides a method for identifying genomic regions which are altered on the chromosomal level and encode genes that are linked by function and are differentially expressed in malignant neoplasia and breast cancer in particular.

15 In yet another embodiment the invention provides the genomic regions 17q21, 3p21 and 12q13 for use in prediction, diagnosis and prognosis as well as prevention and treatment of malignant neoplasia and breast cancer. In particular not only the intragenic regions, but also intergenic regions, pseudogenes or non-transcribed genes of said chromosomal regions can be used for diagnostic, predictive, prognostic and preventive and therapeutic compositions and methods. Therefore sequences of coding or non-coding regions as depicted in this invention are offered by way of illustration and not by way of limitation. As one aspect of this, genomic sequences in between the genomic sequences depicted can be used for similar purposes.

20 In yet another embodiment the invention provides methods of screening for agents which regulate the activity of a polypeptide comprising a polypeptide selected from SEQ ID NO: 27 to 52 and 76 to 98 or encoded by a polynucleotide comprising a polynucleotide selected from SEQ ID NO: 1 to 26 and 53 to 75. A test compound is contacted with a polypeptide comprising a polypeptide selected from SEQ ID NO: 27 to 52 and 76 to 98 or encoded by a polynucleotide comprising a polynucleotide selected from SEQ ID NO: 1 to 26 and 53 to 75. Binding of the test compound to the polypeptide is detected. A test compound which binds to the polypeptide is thereby identified as a potential therapeutic agent for the treatment of malignant neoplasia and more particularly breast cancer.

25 In even another embodiment the invention provides another method of screening for agents which regulate the activity of a polypeptide comprising a polypeptide selected from SEQ ID NO: 27 to 52 and 76 to 98 or encoded by a polynucleotide comprising a polynucleotide selected from SEQ ID NO: 1 to 26 and 53 to 75. A test compound is contacted with a polypeptide comprising a polypeptide selected from SEQ ID NO: 27 to 52 and 76 to 98 or encoded by a polynucleotide comprising a polynucleotide selected from SEQ ID NO: 1 to 26 and 53 to 75. A biological activity

mediated by the polypeptide is detected. A test compound which decreases the biological activity is thereby identified as a potential therapeutic agent for decreasing the activity of the polypeptide encoded by a polypeptide comprising a polypeptide selected from SEQ ID NO: 27 to 52 and 76 to 98 or encoded by a polynucleotide comprising a polynucleotide selected from SEQ ID NO: 1 to 26 and 53 to 75 in malignant neoplasia and breast cancer in particular. A test compound which increases the biological activity is thereby identified as a potential therapeutic agent for increasing the activity of the polypeptide encoded by a polypeptide selected from one of the polypeptides with SEQ ID NO: 27 to 52 and 76 to 98 or encoded by a polynucleotide comprising a polynucleotide selected from SEQ ID NO: 1 to 26 and 53 to 75 in malignant neoplasia and breast cancer in particular.

In another embodiment the invention provides a method of screening for agents which regulate the activity of a polynucleotide comprising a polynucleotide selected from SEQ ID NO: 1 to 26 and 53 to 75. A test compound is contacted with a polynucleotide comprising a polynucleotide selected from SEQ ID NO: 1 to 26 and 53 to 75. Binding of the test compound to the polynucleotide comprising a polynucleotide selected from SEQ ID NO: 1 to 26 and 53 to 75 is detected. A test compound which binds to the polynucleotide is thereby identified as a potential therapeutic agent for regulating the activity of a polynucleotide comprising a polynucleotide selected from SEQ ID NO: 1 to 26 and 53 to 75 in malignant neoplasia and breast cancer in particular.

The invention thus provides polypeptides selected from one of the polypeptides with SEQ ID NO: 27 to 52 and 76 to 98 or encoded by a polynucleotide comprising a polynucleotide selected from SEQ ID NO: 1 to 26 and 53 to 75 which can be used to identify compounds which may act, for example, as regulators or modulators such as agonists and antagonists, partial agonists, inverse agonists, activators, co-activators and inhibitors of the polypeptide comprising a polypeptide selected from SEQ ID NO: 27 to 52 and 76 to 98 or encoded by a polynucleotide comprising a polynucleotide selected from SEQ ID NO: 1 to 26 and 53 to 75. Accordingly, the invention provides reagents and methods for regulating a polypeptide comprising a polypeptide selected from SEQ ID NO: 27 to 52 and 76 to 98 or encoded by a polynucleotide comprising a polynucleotide selected from SEQ ID NO: 1 to 26 and 53 to 75 in malignant neoplasia and more particularly breast cancer. The regulation can be an up- or down regulation. Reagents that modulate the expression, stability or amount of a polynucleotide comprising a polynucleotide selected from SEQ ID NO: 1 to 26 and 53 to 75 or the activity of the polypeptide comprising a polypeptide selected from SEQ ID NO: 27 to 52 and 76 to 98 or encoded by a polynucleotide comprising a polynucleotide selected from SEQ ID NO: 1 to 26 and 53 to 75 can be a protein, a peptide, a peptidomimetic, a nucleic acid, a nucleic acid analogue (e.g. peptide nucleic acid,

locked nucleic acid) or a small molecule. Methods that modulate the expression, stability amount of a polynucleotide comprising a polynucleotide selected from SEQ ID NO: 1 to 26 and 53 to 75 or the activity of the polypeptide comprising a polypeptide selected from SEQ ID NO: 27 to 52 and 76 to 98 or encoded by a polynucleotide comprising a polynucleotide selected from SEQ ID NO: 1 to 26 and 53 to 75 can be gene replacement therapies, antisense, ribozyme and triple nucleic acid approaches.

In one embodiment of the invention provides antibodies which specifically bind to a full-length or partial polypeptide comprising a polypeptide selected from SEQ ID NO: 27 to 52 and 76 to 98 or encoded by a polynucleotide comprising a polynucleotide selected from SEQ ID NO: 1 to 26 and 53 to 75 or a polynucleotide comprising a polynucleotide selected from SEQ ID NO: 1 to 26 and 53 to 75 for use in prediction, prevention, diagnosis, prognosis and treatment of malignant neoplasia and breast cancer in particular.

Yet another embodiment of the invention is the use of a reagent which specifically binds to a polynucleotide comprising a polynucleotide selected from SEQ ID NO: 1 to 26 and 53 to 75 or a polypeptide comprising a polypeptide selected from SEQ ID NO: 27 to 52 and 76 to 98 or encoded by a polynucleotide comprising a polynucleotide selected from SEQ ID NO: 1 to 26 and 53 to 75 in the preparation of a medicament for the treatment of malignant neoplasia and breast cancer in particular.

Still another embodiment is the use of a reagent that modulates the activity or stability of a polypeptide comprising a polypeptide selected from SEQ ID NO: 27 to 52 and 76 to 98 or encoded by a polynucleotide comprising a polynucleotide selected from SEQ ID NO: 1 to 26 and 53 to 75 or the expression, amount or stability of a polynucleotide comprising a polynucleotide selected from SEQ ID NO: 1 to 26 and 53 to 75 in the preparation of a medicament for the treatment of malignant neoplasia and breast cancer in particular.

Still another embodiment of the invention is a pharmaceutical composition which includes a reagent which specifically binds to a polynucleotide comprising a polynucleotide selected from SEQ ID NO: 1 to 26 and 53 to 75 or a polypeptide comprising a polypeptide selected from SEQ ID NO: 27 to 52 and 76 to 98 or encoded by a polynucleotide comprising a polynucleotide selected from SEQ ID NO: 1 to 26 and 53 to 75, and a pharmaceutically acceptable carrier.

Yet another embodiment of the invention is a pharmaceutical composition including a polynucleotide comprising a polynucleotide selected from SEQ ID NO: 1 to 26 and 53 to 75 or

encoding a polypeptide comprising a polypeptide selected from SEQ ID NO: 27 to 52 and 76 to 98.

5 In one embodiment, a reagent which alters the level of expression in a cell of a polynucleotide comprising a polynucleotide selected from SEQ ID NO: 1 to 26 and 53 to 75 or encoding a polypeptide comprising a polypeptide selected from SEQ ID NO: 27 to 52 and 76 to 98, or a sequence complementary thereto, is identified by providing a cell, treating the cell with a test reagent, determining the level of expression in the cell of a polynucleotide comprising a polynucleotide selected from SEQ ID NO: 1 to 26 and 53 to 75 or encoding a polypeptide comprising a polypeptide selected from SEQ ID NO: 27 to 52 and 76 to 98 or a sequence complementary thereto, and comparing the level of expression of the polynucleotide in the treated cell 10 with the level of expression of the polynucleotide in an untreated cell, wherein a change in the level of expression of the polynucleotide in the treated cell relative to the level of expression of the polynucleotide in the untreated cell is indicative of an agent which alters the level of expression of the polynucleotide in a cell.

15 The invention further provides a pharmaceutical composition comprising a reagent identified by this method.

Another embodiment of the invention is a pharmaceutical composition which includes a polypeptide comprising a polypeptide selected from SEQ ID NO: 27 to 52 and 76 to 98 or which is encoded by a polynucleotide comprising a polynucleotide selected from SEQ ID NO: 1 to 26 and 20 53 to 75.

A further embodiment of the invention is a pharmaceutical composition comprising a polynucleotide including a sequence which hybridizes under stringent conditions to a polynucleotide comprising a polynucleotide selected from SEQ ID NO: 1 to 26 and 53 to 75 and encoding a polypeptide exhibiting the same biological function as given for the respective 25 polynucleotide in Table 2 or 3, or encoding a polypeptide comprising a polypeptide selected from SEQ ID NO: 27 to 52 and 76 to 98. Pharmaceutical compositions, useful in the present invention may further include fusion proteins comprising a polypeptide comprising a polynucleotide selected from SEQ ID NO: 27 to 52 and 76 to 98, or a fragment thereof, antibodies, or antibody fragments

BRIEF DESCRIPTION OF THE DRAWINGS

30 Fig. 1 shows a sketch of the chromosome 17 with G-banding pattern and cytogenetic positions. In the blow out at the lower part of the figure a detailed view of the chromosomal area of the long arm of chromosome 17 (17q12-21.1) is provided. Each vertical rectangle depicted

in medium gray, represents a gene as labeled below or above the individual position. The order of genes depicted in this graph has been deduced from experiments questioning the amplification an over expression and from public available data (e.g. UCSC, NCBI or Ensemble).

- 5 Fig. 2 shows the same region as depicted before in Fig. 1 and a cluster representation of the individual expression values measured by DNA-chip hybridization. The gene representing squares are indicated by a dotted line. In the upper part of the cluster representation 4 tumor cell lines, of which two harbor a known HER-2/neu over expression (SKBR3 and AU565), are depicted with their individual expression profiles. Not only the HER-2/neu
- 10 gene shows a clear over expression but as provided by this invention several other genes with in the surrounding. In the middle part of the cluster representation expression data obtained from immune histochemically characterized tumor samples are presented. Two of the depicted probes show a significant over expression of genes marked by the white rectangles. For additional information and comparison expression profiles of several non
- 15 diseased human tissues (rnas obtained from Clontech Inc.) Are provided. Closest relation to the expression profile of HER-2/neu positive tumors displays human brain and neural tissue.
- 20 Fig. 3 provides data from DNA amplification measurements by qpcr (e.g. Taqman). Data indicates that in several analyzed breast cancer cell lines harbor amplification of genes which were located in the previously described region (ARCHEON). Data were displayed for each gene on the x-axis and 40-Ct at the y-axis. Data were normalized to the expression level of GAPDH as seen in the first group of columns.
- 25 Fig. 4 represents a graphical overview on the amplified regions and provides information on the length of the individual amplification and over expression in the analyzed tumor cell lines. The length of the amplification and the composition of genes has a significant impact on the nature of the cancer cell and on the responsiveness on certain drugs, as described elsewhere.

DETAILED DESCRIPTION OF THE INVENTION

DEFINITIONS

- 30 "Differential expression", as used herein, refers to both quantitative as well as qualitative differences in the genes' expression patterns depending on differential development and/or tumor growth. Differentially expressed genes may represent "marker genes," and/or "target genes". The

expression pattern of a differentially expressed gene disclosed herein may be utilized as part of a prognostic or diagnostic breast cancer evaluation. Alternatively, a differentially expressed gene disclosed herein may be used in methods for identifying reagents and compounds and uses of these reagents and compounds for the treatment of breast cancer as well as methods of treatment.

5 "Biological activity" or "bioactivity" or "activity" or "biological function", which are used interchangeably, herein mean an effector or antigenic function that is directly or indirectly performed by a polypeptide (whether in its native or denatured conformation), or by any fragment thereof *in vivo* or *in vitro*. Biological activities include but are not limited to binding to polypeptides, binding to other proteins or molecules, enzymatic activity, signal transduction,
10 activity as a DNA binding protein, as a transcription regulator, ability to bind damaged DNA, etc. A bioactivity can be modulated by directly affecting the subject polypeptide. Alternatively, a bioactivity can be altered by modulating the level of the polypeptide, such as by modulating expression of the corresponding gene.

15 The term "marker" or "biomarker" refers a biological molecule, e.g., a nucleic acid, peptide, hormone, etc., whose presence or concentration can be detected and correlated with a known condition, such as a disease state.

"Marker gene," as used herein, refers to a differentially expressed gene which expression pattern may be utilized as part of predictive, prognostic or diagnostic malignant neoplasia or breast cancer evaluation, or which, alternatively, may be used in methods for identifying compounds useful for
20 the treatment or prevention of malignant neoplasia and breast cancer in particular. A marker gene may also have the characteristics of a target gene.

"Target gene", as used herein, refers to a differentially expressed gene involved in breast cancer
a manner by which modulation of the level of target gene expression or of target gene product activity may act to ameliorate symptoms of malignant neoplasia and breast cancer in particular. A
25 target gene may also have the characteristics of a marker gene.

The term "biological sample", as used herein, refers to a sample obtained from an organism or from components (e.g., cells) of an organism. The sample may be of any biological tissue or fluid. Frequently the sample will be a "clinical sample" which is a sample derived from a patient. Such samples include, but are not limited to, sputum, blood, blood cells (e.g., white cells), tissue or fine
30 needle biopsy samples, cell-containing bodyfluids, free floating nucleic acids, urine, peritoneal fluid, and pleural fluid, or cells therefrom. Biological samples may also include sections of tissues such as frozen sections taken for histological purposes.

By "array" or "matrix" is meant an arrangement of addressable locations or "addresses" on a device. The locations can be arranged in two dimensional arrays, three dimensional arrays, other matrix formats. The number of locations can range from several to at least hundreds of thousands. Most importantly, each location represents a totally independent reaction site. Arrays include but are not limited to nucleic acid arrays, protein arrays and antibody arrays. A "nucleic acid array" refers to an array containing nucleic acid probes, such as oligonucleotide polynucleotides or larger portions of genes. The nucleic acid on the array is preferably single stranded. Arrays wherein the probes are oligonucleotides are referred to as "oligonucleotide arrays" or "oligonucleotide chips." A "microarray," herein also refers to a "biochip" or "biological chip", an array of regions having a density of discrete regions of at least about 100/cm², and preferably at least about 1000/cm². The regions in a microarray have typical dimensions, e.g. diameters, in the range of between about 10-250 μ m, and are separated from other regions in the array by about the same distance. A "protein array" refers to an array containing polypeptide probes or protein probes which can be in native form or denatured. An "antibody array" refers to an array containing antibodies which include but are not limited to monoclonal antibodies (e.g. from a mouse), chimeric antibodies, humanized antibodies or phage antibodies and single chain antibodies as well as fragments from antibodies.

The term "agonist", as used herein, is meant to refer to an agent that mimics or upregulates (e.g., potentiates or supplements) the bioactivity of a protein. An agonist can be a wild-type protein or derivative thereof having at least one bioactivity of the wild-type protein. An agonist can also be a compound that upregulates expression of a gene or which increases at least one bioactivity of a protein. An agonist can also be a compound which increases the interaction of a polypeptide with another molecule, e.g., a target peptide or nucleic acid.

The term "antagonist" as used herein is meant to refer to an agent that downregulates (e.g., suppresses or inhibits) at least one bioactivity of a protein. An antagonist can be a compound which inhibits or decreases the interaction between a protein and another molecule, e.g., a target peptide, a ligand or an enzyme substrate. An antagonist can also be a compound that downregulates expression of a gene or which reduces the amount of expressed protein present.

"Small molecule" as used herein, is meant to refer to a composition, which has a molecular weight of less than about 5 kD and most preferably less than about 4 kD. Small molecules can be nucleic acids, peptides, polypeptides, peptidomimetics, carbohydrates, lipids or other organic (carbon-containing) or inorganic molecules. Many pharmaceutical companies have extensive libraries of chemical and/or biological mixtures, often fungal, bacterial, or algal extracts, which can be screened with any of the assays of the invention to identify compounds that modulate a bioactivity.

The terms "modulated" or "modulation" or "regulated" or "regulation" and "differentially regulated" as used herein refer to both upregulation (i.e., activation or stimulation (e.g., by agonizing or potentiating) and down regulation [i.e., inhibition or suppression (e.g., by antagonizing, decreasing or inhibiting)]).

5 "Transcriptional regulatory unit" refers to DNA sequences, such as initiation signals, enhancers, and promoters, which induce or control transcription of protein coding sequences with which they are operably linked. In preferred embodiments, transcription of one of the genes is under the control of a promoter sequence (or other transcriptional regulatory sequence) which controls the expression of the recombinant gene in a cell-type in which expression is intended. It will also be
10 understood that the recombinant gene can be under the control of transcriptional regulatory sequences which are the same or which are different from those sequences which control transcription of the naturally occurring forms of the polypeptide.

The term "derivative" refers to the chemical modification of a polypeptide sequence, or a polynucleotide sequence. Chemical modifications of a polynucleotide sequence can include, for
15 example, replacement of hydrogen by an alkyl, acyl, or amino group. A derivative polynucleotide encodes a polypeptide which retains at least one biological or immunological function of the natural molecule. A derivative polypeptide is one modified by glycosylation, pegylation, or any similar process that retains at least one biological or immunological function of the polypeptide from which it was derived.

20 The term "nucleotide analog" refers to oligomers or polymers being at least in one feature different from naturally occurring nucleotides, oligonucleotides or polynucleotides, but exhibiting functional features of the respective naturally occurring nucleotides (e.g. base pairing, hybridization, coding information) and that can be used for said compositions. The nucleotide
25 analogs can consist of non-naturally occurring bases or polymer backbones, examples of which are LNAs, PNAs and Morpholinos. The nucleotide analog has at least one molecule different from its naturally occurring counterpart or equivalent.

"BREAST CANCER GENES" or "BREAST CANCER GENE" as used herein refers to the polynucleotides of SEQ ID NO: 1 to 26 and 53 to 75, as well as derivatives, fragments, analogs and homologues thereof, the polypeptides encoded thereby, the polypeptides of SEQ ID NO: 27 to
30 52 and 76 to 98 as well as derivatives, fragments, analogs and homologues thereof and the corresponding genomic transcription units which can be derived or identified with standard techniques well known in the art using the information disclosed in Tables 1 to 5 and Figures 1 to 4. The GenBank, Locuslink ID and the UniGene accession numbers of the polynucleotide

sequences of the SEQ ID NO: 1 to 26 and 53 to 75 and the polypeptides of the SEQ ID NO: 252 and 76 to 98 are shown in Table 1, the gene description, gene function and subcellular localization is given in Tables 2 and 3.

5 The term "chromosomal region" as used herein refers to a consecutive DNA stretch on a chromosome which can be defined by cytogenetic or other genetic markers such as e.g. restriction length polymorphisms (RFLPs), single nucleotide polymorphisms (SNPs), expressed sequence tags (ESTs), sequence tagged sites (STSs), microsatellites, variable number of tandem repeats (VNTRs) and genes. Typically a chromosomal region consists of up to 2 Megabases (MB), up to 4 MB, up to 6 MB, up to 8 MB, up to 10 MB, up to 20 MB or even more MB.

10 The term "altered chromosomal region" or "abberant chromosomal region" refers to a structural change of the chromosomal composition and DNA sequence, which can occur by the following events: amplifications, deletions, inversions, insertions, translocations and/or viral integration. Polyploidy, where a given cell harbors more than two copies of a chromosome, is within the meaning of the term "amplification" of a chromosome or chromosomal region.

15 The present invention provides polynucleotide sequences and proteins encoded thereby, as well as probes derived from the polynucleotide sequences, antibodies directed to the encoded proteins, and their predictive, preventive, diagnostic, prognostic and therapeutic uses for individuals which are at risk for or which have malignant neoplasia and breast cancer in particular. The sequences disclosed herein have been found to be differentially expressed in samples from breast cancer.

20 The present invention is based on the identification of 43 genes that are differentially regulated (up- or downregulated) in tumor biopsies of patients with clinical evidence of breast cancer. The identification of 43 human genes which were not known to be differentially regulated in breast cancer states and their significance for the disease is described in the working examples hereinafter. The characterization of the co-expression of these genes provides newly identified roles in breast cancer. The gene names, the database accession numbers (GenBank and UniGene) as well as putative or known functions of the encoded proteins and their subcellular localization are given in Tables 1 to 4. The primer sequences used for the gene amplification are shown in Table 5.

30 In either situation, detecting expression of these genes in excess or in with lower level compared to normal expression provides the basis for the diagnosis of malignant neoplasia and breast cancer. Furthermore, in testing the efficacy of compounds during clinical trials, a decrease in the level of the expression of these genes corresponds to a return from a disease condition to a normal state, and thereby indicates a positive effect of the compound.

Another aspect of the present invention is based on the observation that neighboring genes within defined genomic regions functionally interact and influence each others function directly or indirectly. A genomic region encoding functionally interacting genes that are co-amplified and co-expressed in neoplastic lesions has been defined as an "ARCHEON". (ARCHEON = Altered
5 Region of Changed Chromosomal Expression Observed in Neoplasms). Chromosomal alterations often affect more than one gene. This is true for amplifications, duplications, insertions, integrations, inversions, translocations, and deletions. These changes can have influence on the expression level of single or multiple genes. Most commonly in the field of cancer diagnostics and treatment the changes of expression levels have been investigated for single, putative relevant
10 target genes such as MLV12 (5p14), NRASL3 (6p12), EGFR (7p12), c-myc (8q23), Cyclin D1 (11q13), IGF1R (15q25), HER-2/neu (17q21), PCNA (20q12). However, the altered expression level and interaction of multiple (i.e. more than two) genes within one genomic region with each other has not been addressed. Genes of an ARCHEON form gene clusters with tissue specific expression patterns. The mode of interaction of individual genes within such a gene cluster suspected to represent an ARCHEON can be either protein-protein or protein-nucleic acid
15 interaction, which may be illustrated but not limited by the following examples: ARCHEON gene interaction may be in the same signal transduction pathway, may be receptor to ligand binding, receptor kinase and SH2 or SH3 binding, transcription factor to promoter binding, nuclear hormone receptor to transcription factor binding, phosphogroup donation (e.g. kinases) and acceptance (e.g. phosphoprotein), mRNA stabilizing protein binding and transcriptional processes.
20 The individual activity and specificity of a pair genes and or the proteins encoded thereby or of a group of such in a higher order, may be readily deduced from literature, published or deposited within public databases by the skilled person. However in the context of an ARCHEON the interaction of members being part of an ARCHEON will potentiate, exaggerate or reduce their
25 singular functions. This interaction is of importance in defined normal tissues in which they are normally co-expressed. Therefore, these clusters have been commonly conserved during evolution. The aberrant expression of members of these ARCHEON in neoplastic lesions, however, (especially within tissues in which they are normally not expressed) has influence on tumor characteristics such as growth, invasiveness and drug responsiveness. Due to the interaction
30 of these neighboring genes it is of importance to determine the members of the ARCHEON which are involved in the deregulation events. In this regard amplification and deletion events in neoplastic lesions are of special interest.

The invention relates to a method for the detection of chromosomal alterations by (a) determining the relative mRNA abundance of individual mRNA species or (b) determining the copy number of
35 one or more chromosomal region(s) by quantitative PCR. In one embodiment information on the

genomic organization and spatial regulation of chromosomal regions is assessed by bioinformatic analysis of the sequence information of the human genome (UCSC, NCBI) and then combined with RNA expression data from GeneChip™ DNA-Arrays (Affymetrix) and/or quantitative (TaqMan) from RNA-samples or genomic DNA.

- 5 In a further embodiment the functional relationship of genes located on a chromosomal region which is altered (amplified or deleted) is established. The altered chromosomal region is defined as an ARCHEON if genes located on that region functionally interact.

The 17q21 locus was investigated as one model system, harboring the HER-2/neu gene. By establishing a high-resolution assay to detect amplification events in neighboring genes, 43 genes that are commonly co-amplified in breast cancer cell lines and patient samples were identified. Using gene array technologies and immunological methods their co-overexpression in tumor samples was demonstrated. Surprisingly, by clustering tissue samples with HER-2/neu positive tumor samples, it was found that the expression pattern of this larger genomic region (consisting of 10 genes) is very similar to control brain tissue. HER-2/neu negative breast tumor tissue did not show a similar expression pattern. Indeed, some of the genes within these clusters are important for neural development (HER-2/neu, THRA) in mouse model systems or are described to be expressed in neural cells (NeuroD2). Moreover, by searching for similar gene combinations in human and rodent genome additional homologous chromosomal regions on chromosome 3p21 and 12q13 harboring several isoforms of the respective genes (see below) were found. There was strong evidence for multiple interactions between the 43 candidate genes, as being part of identical pathways (HER-2, neu, GRB7, CrkRS, CDC6), influencing the expression of each other (HER-2/neu, THRA, RARA), interacting with each other (PPARGBP, THRA, RARA, NR1D1 or HER-2/neu, GRB7) or expressed in defined tissues (CACNB1, PPARGBP, etc.). Interestingly, genomic regions of the ARCHEONS that were identified are amplified in acquired Tamoxifen resistance of HER-2/neu negative cells (MCF7), which are normally sensitive to Tamoxifen treatment [Achuthan et al., 2001,(2)].

Moreover, altered responsiveness to treatment due to the alterations of the genes within the ARCHEONS was observed. Surprisingly, genes within the ARCHEONS are of importance even in the absence of HER-2/neu homologues. Some of the genes within the ARCHEONS, do not serve as marker genes for prognostic purposes, but have already been known as targets for therapeutic intervention. For example TOP2 alpha is a target of anthracyclins. THRA and RARA can be targeted by hormones and hormone analogs (e.g. T3, rT3, RA). Due to their high affinity binding sites and available screening assays (reporter assays based on their transcriptional potential) the hormone receptors which are shown to be linked to neoplastic pathophysiology

the first time herein are ideal targets for drug screening and treatment of malignant neoplasia and breast cancer in particular. In this regard it is essential to know which members of the ARCHEON are altered in the neoplastic lesions. Particularly it is important to know the nature, number and extent to which the ARCHEON genes are amplified or deleted. The ARCHEONs are flanked by similar, endogenous retroviruses (e.g. HERV-K= "human endogenous retrovirus"), some of which are activated in breast cancer. These viruses may have also been involved in the evolutionary duplication of the ARCHEONs.

The analysis of the 17q21 region proved data obtained by IHC and identified several additional genes being co-amplified with the HER-2/neu gene. Comparative Analysis of RNA-based quantitative RT-PCR (TaqMan) with DNA-based qPCR from tumor cell lines identified the same amplified region. Genes at the 17q11.2 -21. region are offered by way of illustration not by way of limitation. A graphical display of the described chromosomal region is provided in Figure 1.

Biological relevance of the genes which are part of the 17q21 ARCHEON

MLN50

By differential screening of cDNAs from breast cancer-derived metastatic axillary lymph nodes, TRAF4 and 3 other novel genes (MLN51, MLN62, MLN64) were identified that are overexpressed in breast cancer [Tomasetto et al., 1995, (3)]. One gene, which they designated MLN50, was mapped to 17q11-q21.3 by radioactive in situ hybridization. In breast cancer cell lines, overexpression of the 4 kb MLN50 mRNA was correlated with amplification of the gene and with amplification and overexpression of ERBB2, which maps to the same region. The authors suggested that the 2 genes belong to the same amplicon. Amplification of chromosomal region 17q11-q21 is one of the most common events occurring in human breast cancers. They report that the predicted 261-amino acid MLN50 protein contains an N-terminal LIM domain and a C-terminal SH3 domain. They renamed the protein LASP1, for 'LIM and SH3 protein.' Northern blot analysis revealed that LASP1 mRNA was expressed at a basal level in all normal tissues examined and overexpressed in 8% of primary breast cancers. In most of these cancers, LASP1 and ERBB2 were simultaneously overexpressed.

MLLT6

The MLLT6 (AF17) gene encodes a protein of 1,093 amino acids, containing a leucine-zipper dimerization motif located 3-prime of the fusion point and a cysteine-rich domain at the end terminus. AF17 was found to contain stretches of amino acids previously associated with domains involved in transcriptional repression or activation.

Chromosome translocations involving band 11q23 are associated with approximately 10 patients with acute lymphoblastic leukemia (ALL) and more than 5% of patients with myeloid leukemia (AML). The gene at 11q23 involved in the translocations is vari-
 designated ALL1, HRX, MLL, and TRX1. The partner gene in one of the rarer translocat-
 5 t(11;17)(q23;q21), designated MLLT6 on 17q12.

ZNF144 (Mel18)

Mel18 cDNA encodes a novel cys-rich zinc finger motif. The gene is expressed strongly in tumor cell lines, but its normal tissue expression was limited to cells of neural origin and especially abundant in fetal neural cells. It belongs to a RING-finger motif family which inc-
 10 BMI1. The MEL18/BMI1 gene family represents a mammalian homolog of the Drosophila 'polycomb' gene group, thereby belonging to a memory mechanism involved in maintaining the expression pattern of key regulatory factors such as Hox genes. Bmi1, Mel18 and M33 genes as representative examples of mouse Pc-G genes. Common phenotypes observed in knockout mutant for each of these genes indicate an important role for Pc-G genes not only in regulation
 15 Hox gene expression and axial skeleton development but also in control of proliferation and survival of haematopoietic cell lineages. This is in line with the observed proliferation and differentiation deregulation observed in lymphoblastic leukemia. The MEL18 gene is conserved among vertebrates. Its mRNA is expressed at high levels in placenta, lung, and kidney, and at lower levels in liver, pancreas, and skeletal muscle. Interestingly, cervical and lumbo-sacral-HOX gene expression is altered in several primary breast cancers with respect to normal breast tissue with
 20 HoxB gene cluster being present on 17q distal to the 17q21 locus. Moreover, delayed differentiation with persistent nests of proliferating cells was found in endothelial cells cocultured with HOXB7-transduced SkBr3 cells, which exhibit a 17q21 amplification. Tumorigenicity of these cells has been evaluated in vivo. Xenograft in athymic nude mice showed that
 25 SkBr3/HOXB7 cells developed tumors with an increased number of blood vessels, even if irradiated or not, whereas parental SkBr3 cells did not show any tumor take unless mice were sublethally irradiated. As part of this invention, we have found MEL18 to be overexpressed specifically in tumors bearing Her-2/neu gene amplification, which can be critical for tumor progression.

PHOSPHATIDYLINOSITOL-4-PHOSPHATE 5-KINASE, TYPE II, BETA; PIP5K2B

Phosphoinositide kinases play central roles in signal transduction. Phosphatidylinositol 4-phosphate 5-kinases (PIP5Ks) phosphorylate phosphatidylinositol 4-phosphate, giving rise to phosphatidylinositol 4,5-bisphosphate. The PIP5K enzymes exist as multiple isoforms that

various immunoreactivities, kinetic properties, and molecular masses. They are unique in that they possess almost no homology to the kinase motifs present in other phosphatidylinositol, protein, and lipid kinases. By screening a human fetal brain cDNA library with the PIP5K2B EST the full length gene could be isolated. The deduced 416-amino acid protein is 78% identical to PIP5K2A. Using SDS-PAGE, the authors estimated that bacterially expressed PIP5K2B has a molecular mass of 47 kD. Northern blot analysis detected a 6.3-kb PIP5K2B transcript which was abundantly expressed in several human tissues. PIP5K2B interacts specifically with the juxtamembrane region of the p55 TNF receptor (TNFR1) and PIP5K2B activity is increased in mammalian cells by treatment with TNF-alpha. A modeled complex with membrane-bound substrate and ATP shows how a phosphoinositide kinase can phosphorylate its substrate in situ at the membrane interface. The substrate-binding site is open on 1 side, consistent with dual specificity for phosphatidylinositol 3- and 5-phosphates. Although the amino acid sequence of PIP5K2A does not show homology to known kinases, recombinant PIP5K2A exhibited kinase activity. PIP5K2A contains a putative Src homology 3 (SH3) domain-binding sequence. Overexpression of mouse PIP5K1B in COS7 cells induced an increase in short actin fibers and a decrease in actin stress fibers.

TEM7

Using serial analysis of gene expression (SAGE) a partial cDNAs corresponding to several tumor endothelial markers (TEMs) that displayed elevated expression during tumor angiogenesis could be identified. Among the genes identified was TEM7. Using database searches and 5-prime RACE the entire TEM7 coding region, which encodes a 500-amino acid type I transmembrane protein, has been described. The extracellular region of TEM7 contains a plexin-like domain and has weak homology to the ECM protein nidogen. The function of these domains, which are usually found secreted and extracellular matrix molecules, is unknown. Nidogen itself belongs to the entactin protein family and helps to determine pathways of migrating axons by switching from circumferential to longitudinal migration. Entactin is involved in cell migration, as it promotes trophoblast outgrowth through a mechanism mediated by the RGD recognition site, and plays an important role during invasion of the endometrial basement membrane at implantation. As entactin promotes thymocyte adhesion but affects thymocyte migration only marginally, it is suggested that entactin may play a role in thymocyte localization during T cell development.

In situ hybridization analysis of human colorectal cancer demonstrated that TEM7 was expressed clearly in the endothelial cells of the tumor stroma but not in the endothelial cells of normal colonic tissue. Using in situ hybridization to assay expression in various normal adult mouse

tissues, they observed that TEM7 was largely undetectable in mouse tissues or tumors, but was abundantly expressed in mouse brain.

ZNFN1A3

By screening a B-cell cDNA library with a mouse Aiolos N-terminal cDNA probe, a cDNA
5 encoding human Aiolos, or ZNFN1A3, was obtained. The deduced 509-amino acid protein, which
is 86% identical to its mouse counterpart, has 4 DNA-binding zinc fingers in its N terminus and 2
zinc fingers that mediate protein dimerization in its C terminus. These domains are 100% and 96%
homologous to the corresponding domains in the mouse protein, respectively. Northern blot
analysis revealed strong expression of a major 11.0- and a minor 4.4-kb ZNFN1A3 transcript in
10 peripheral blood leukocytes, spleen, and thymus, with lower expression in liver, small intestine,
and lung.

Ikaros (ZNFN1A1), a hemopoietic zinc finger DNA-binding protein, is a central regulator of
lymphoid differentiation and is implicated in leukemogenesis. The execution of normal function of
Ikaros requires sequence-specific DNA binding, transactivation, and dimerization domains. Mice
15 with a mutation in a related zinc finger protein, Aiolos, are prone to B-cell lymphoma. In
chemically induced murine lymphomas allelic losses on markers surrounding the *Znfn1a1* gene
were detected in 27% of the tumors analyzed. Moreover specific Ikaros expression was in primary
mouse hormone-producing anterior pituitary cells and substantial for Fibroblast growth factor
receptor 4 (FGFR4) expression, which itself is implicated in a multitude of endocrine cell
20 hormonal and proliferative properties with FGFR4 being differentially expressed in normal and
neoplastic pituitary. Moreover Ikaros binds to chromatin remodelling complexes containing
SWI/SNF proteins, which antagonize Polycomb function. Interestingly at the telomeric end of the
disclosed ARCHEON the SWI/SNF complex member SMARCE1 (= SWI/SNF-related, matrix-
associated, actin-dependent regulators of chromatin) is located and part of the described
25 amplification. Due to the related binding specificities of Ikaros and Palindrom Binding Protein
(PBP) it is suggestive, that ZNFN1A3 is able to regulate the Her-2/neu enhancer.

PPP1R1B

Midbrain dopaminergic neurons play a critical role in multiple brain functions, and abnormal
signaling through dopaminergic pathways has been implicated in several major neurologic and
30 psychiatric disorders. One well-studied target for the actions of dopamine is DARPP32. In the
densely dopamine- and glutamate-innervated rat caudate-putamen, DARPP32 is expressed in
medium-sized spiny neurons that also express dopamine D1 receptors. The function of DARPP32

seems to be regulated by receptor stimulation. Both dopaminergic and glutamatergic (NMDA) receptor stimulation regulate the extent of DARPP32 phosphorylation, but in opposite directions.

The human DARPP32 was isolated from a striatal cDNA library. The 204-amino acid DARPP32 protein shares 88% and 85% sequence identity, respectively, with bovine and rat DARPP32 proteins. The DARPP32 sequence is particularly conserved through the N terminus, which represents the active portion of the protein. Northern blot analysis demonstrated that the 2.1-kb DARPP32 mRNA is more highly expressed in human caudate than in cortex. In situ hybridization to postmortem human brain showed a low level of DARPP32 expression in all neocortical layers, with the strongest hybridization in the superficial layers. CDK5 phosphorylated DARPP32 in vitro and in intact brain cells. Phospho-thr75 DARPP32 inhibits PKA in vitro by a competitive mechanism. Decreasing phospho-thr75 DARPP32 in striatal cells either by a CDK5-specific inhibitor or by using genetically altered mice resulted in increased dopamine-induced phosphorylation of PKA substrates and augmented peak voltage-gated calcium currents. Thus, DARPP32 is a bifunctional signal transduction molecule which, by distinct mechanisms, controls a serine/threonine kinase and a serine/threonine phosphatase.

DARPP32 and t-DARPP are overexpressed in gastric cancers. It's suggested that overexpression of these 2 proteins in gastric cancers may provide an important survival advantage to neoplastic cells. It could be demonstrated that Darpp32 is an obligate intermediate in progesterone-facilitated sexual receptivity in female rats and mice. The facilitative effect of progesterone on sexual receptivity in female rats was blocked by antisense oligonucleotides to Darpp32. Homozygous mice carrying a null mutation for the Darpp32 gene exhibited minimal levels of progesterone-facilitated sexual receptivity when compared to their wildtype littermates, and progesterone significantly increased hypothalamic cAMP levels and cAMP-dependent protein kinase activity.

CACNB1

In 1991 a cDNA clone encoding a protein with high homology to the beta subunit of the rabbit skeletal muscle dihydropyridine-sensitive calcium channel from a rat brain cDNA library [Pragnell et al., 1991, (4)]. This rat brain beta-subunit cDNA hybridized to a 3.4-kb message that was expressed in high levels in the cerebral hemispheres and hippocampus and much lower levels in cerebellum. The open reading frame encodes 597 amino acids with a predicted mass of 65,679 Da which is 82% homologous with the skeletal muscle beta subunit. The corresponding human beta-subunit gene was localized to chromosome 17 by analysis of somatic cell hybrids. The authors suggested that the encoded brain beta subunit, which has a primary structure highly similar to its

isoform in skeletal muscle, may have a comparable role as an integral regulatory component of neuronal calcium channel.

RPL19

The ribosome is the only organelle conserved between prokaryotes and eukaryotes. In eukaryote
 5 this organelle consists of a 60S large subunit and a 40S small subunit. The mammalian ribosome
 contains 4 species of RNA and approximately 80 different ribosomal proteins, most of which
 appear to be present in equimolar amounts. In mammalian cells, ribosomal proteins can account
 for up to 15% of the total cellular protein, and the expression of the different ribosomal protein
 genes, which can account for up to 7 to 9% of the total cellular mRNAs, is coordinately regulated
 10 to meet the cell's varying requirements for protein synthesis. The mammalian ribosomal protein
 genes are members of multigene families, most of which are composed of multiple processed
 pseudogenes and a single functional intron-containing gene. The presence of multiple pseudogenes
 hampered the isolation and study of the functional ribosomal protein genes. By study of somatic
 cell hybrids, it has been elucidated that DNA sequences complementary to 6 mammalian
 15 ribosomal protein cDNAs could be assigned to chromosomes 5, 8, and 17. Ten fragments mapped
 to 3 chromosomes [Nakamichi et al., 1986, (5)]. These are probably a mixture of functional
 (expressed) genes and pseudogenes. One that maps to 5q23-q33 rescues Chinese hamster emetine-
 resistance mutations in interspecies hybrids and is therefore the transcriptionally active RPS14
 gene. In 1989 a PCR-based strategy for the detection of intron-containing genes in the presence of
 20 multiple pseudogenes was described. This technique was used to identify the intron-containing
 PCR products of 7 human ribosomal protein genes and to map their chromosomal locations by
 hybridization to human/rodent somatic cell hybrids [Feo et al., 1992, (6)]. All 7 ribosomal protein
 genes were found to be on different chromosomes: RPL19 on 17p12-q11; RPL30 on 8; RPL35A on
 18; RPL36A on 14; RPS6 on 9pter-p13; RPS11 on 19cen-qter; and RPS17 on 11pter-p13. These
 25 are also different sites from the chromosomal location of previously mapped ribosomal protein
 genes S14 on chromosome 5, S4 on Xq and Yp, and RP117A on 9q3-q34. By fluorescence in situ
 hybridization the position of the RPL19 gene was mapped to 17q11 [Davies et al., 1989, (7)].

PPARBP, PBP, CRSP1, CRSP200, TRIP2, TRAP220, RB18A, DRIP230

The thyroid hormone receptors (TRs) are hormone-dependent transcription factors that regulate
 30 expression of a variety of specific target genes. They must specifically interact with a number of
 proteins as they progress from their initial translation and nuclear translocation to
 heterodimerization with retinoid X receptors (RXRs), functional interactions with other
 transcription factors and the basic transcriptional apparatus, and eventually, degradation. To help

elucidate the mechanisms that underlie the transcriptional effects and other potential functions of TRs, the yeast interaction trap, a version of the yeast 2-hybrid system, was used to identify proteins that specifically interact with the ligand-binding domain of rat TR-beta-1 (THRB) [Lee et al., 1995, (8)]. The authors isolated HeLa cell cDNAs encoding several different TR-interacting proteins (TRIPs), including TRIP2. TRIP2 interacted with rat Thrb only in the presence of thyroid hormone. It showed a ligand-independent interaction with RXR-alpha, but did not interact with the glucocorticoid receptor (NR3C1) under any condition. By immunoscreening a human B-lymphoma cell cDNA expression library with the anti-p53 monoclonal antibody PAb1801, PPARBP was identified, which was called RB18A for 'recognized by PAb1801 monoclonal antibody' [Drane et al., 1997, (9)]. The predicted 1,566-amino acid RB18A protein contains several potential nuclear localization signals, 13 potential N-glycosylation sites, and a high number of potential phosphorylation sites. Despite sharing common antigenic determinants with p53, RB18A does not show significant nucleotide or amino acid sequence similarity with p53. Whereas the calculated molecular mass of RB18A is 166 kD, the apparent mass of recombinant RB18A was 205 kD by SDS-PAGE analysis. The authors demonstrated that RB18A shares functional properties with p53, including DNA binding, p53 binding, and self-oligomerization. Furthermore, RB18A was able to activate the sequence-specific binding of p53 to DNA, which was induced through an unstable interaction between both proteins. Northern blot analysis of human tissues detected an 8.5-kb RB18A transcript in all tissues examined except kidney, with highest expression in heart. Moreover mouse Pparbp, which was called Pbp for 'Ppar-binding protein,' as a protein that interacts with the Ppar-gamma (PPARG) ligand-binding domain in a yeast 2-hybrid system was identified [Zhu et al., 1997, (10)]. The authors found that Pbp also binds to PPAR-alpha (PPARA), RAR-alpha (RARA), RXR, and TR-beta-1 in vitro. The binding of Pbp to these receptors increased in the presence of specific ligands. Deletion of the last 12 amino acids from the C terminus of PPAR-gamma resulted in the abolition of interaction between Pbp and PPAR-gamma. Pbp modestly increased the transcriptional activity of PPAR-gamma, and a truncated form of Pbp acted as a dominant-negative repressor, suggesting that Pbp is a genuine transcriptional co-activator for PPAR. The predicted 1,560-amino acid Pbp protein contains 2 LXXLL motifs, which are considered necessary and sufficient for the binding of several co-activators to nuclear receptors. Northern blot analysis detected Pbp expression in all mouse tissues examined, with higher levels in liver, kidney, lung, and testis. In situ hybridization showed that Pbp is expressed during mouse ontogeny, suggesting a possible role for Pbp in cellular proliferation and differentiation. In adult mouse, in situ hybridization detected Pbp expression in liver, bronchial epithelium in the lung, intestinal mucosa, kidney cortex, thymic cortex, splenic follicles, and seminiferous epithelium in testis. Later on PPARBP was identified, which was called

TRAP220, from an immunopurified TR-alpha (THRA)-TRAP complex [Yuan et al., 1998, (11)]. The authors cloned Jurkat cell cDNAs encoding TRAP220. The predicted 1,581-amino acid TRAP220 protein contains LXXLL domains, which are found in other nuclear receptor-interacting proteins. TRAP220 is nearly identical to RB18A, with these proteins differing primarily by an extended N terminus on TRAP220. In the absence of TR-alpha, TRAP220 appears to reside in a single complex with other TRAPs. TRAP220 showed a direct ligand-dependent interaction with TR-alpha, which was mediated through the C terminus of TR-alpha and, at least in part, the LXXLL domains of TRAP220. TRAP220 also interacted with other nuclear receptors, including vitamin D receptor, RARA, RXRA, PPARA, PPARG, and estrogen receptor-alpha (ESR1; 133430), in a ligand-dependent manner. TRAP220 moderately stimulated human TR-alpha-mediated transcription in transfected cells, whereas a fragment containing the LXXLL motifs acted as a dominant-negative inhibitor of nuclear receptor-mediated transcription both in transfected cells and in cell-free transcription systems. Further studies indicated that TRAP220 plays a major role in anchoring other TRAPs to TR-alpha during the function of the TR-alpha-TRAP complex and that TRAP220 may be a global co-activator for the nuclear receptor superfamily. PBP, a nuclear receptor co-activator, interacts with estrogen receptor-alpha (ESR1) in the absence of estrogen. This interaction was enhanced in the presence of estrogen, but was reduced in the presence of the anti-estrogen Tamoxifen. Transfection of PBP into cultured cells resulted in enhancement of estrogen-dependent transcription, indicating that PBP serves as a co-activator in estrogen receptor signaling. To examine whether overexpression of PBP plays a role in breast cancer because of its co-activator function in estrogen receptor signaling, the levels of PBP expression in breast tumors was determined [Zhu et al., 1999, (12)]. High levels of PBP expression were detected in approximately 50% of primary breast cancers and breast cancer cell lines by ribonuclease protection analysis, in situ hybridization, and immunoperoxidase staining. By using FISH, the authors mapped the PBP gene to 17q12, a region that is amplified in some breast cancers. They found PBP gene amplification in approximately 24% (6 of 25) of breast tumors and approximately 30% (2 of 6) of breast cancer cell lines, implying that PBP gene overexpression can occur independent of gene amplification. They determined that the PBP gene comprises 17 exons that together span more than 37 kb. Their findings, in particular PBP gene amplification, suggested that PBP, by its ability to function as an estrogen receptor-alpha co-activator, may play a role in mammary epithelial differentiation and in breast carcinogenesis.

NEUROD2

Basic helix-loop-helix (bHLH) proteins are transcription factors involved in determining cell type during development. In 1995 a bHLH protein was described, termed NeuroD (for 'neurogenic

differentiation'), that functions during neurogenesis [Lee et al., 1995, (13)]. The human NEUROD gene maps to chromosome 2q32. The cloning and characterization of 2 additional NEUROD genes, NEUROD2 and NEUROD3 was described in 1996 [McCormick et al., 1996, (14)]. Sequences for the mouse and human homologues were presented. NEUROD2 shows a high degree of homology to the bHLH region of NEUROD, whereas NEUROD3 is more distantly related. The authors found that mouse neuroD2 was initially expressed at embryonic day 11, with persistent expression in the adult nervous system. Similar to neuroD, neuroD2 appears to mediate neuronal differentiation. The human NEUROD2 was mapped to 17q12 by fluorescence in situ hybridization and the mouse homologue to chromosome 11 [Tamimi et al., 1997, (15)].

10 TELETHONIN

Telethonin is a sarcomeric protein of 19 kD found exclusively in striated and cardiac muscle. It appears to be localized to the Z disc of adult skeletal muscle and cultured myocytes. Telethonin is a substrate of titin, which acts as a molecular 'ruler' for the assembly of the sarcomere by providing spatially defined binding sites for other sarcomeric proteins. After activation by phosphorylation and calcium/calmodulin binding, titin phosphorylates the C-terminal domain of telethonin in early differentiating myocytes. The telethonin gene has been mapped to 17q12, adjacent to the phenylethanolamine N-methyltransferase gene [Valle et al., 1997, (16)].

PENT, PNMT

Phenylethanolamine N-methyltransferase catalyzes the synthesis of epinephrine from norepinephrine, the last step of catecholamine biosynthesis. The cDNA clone was first isolated in 1998 for bovine adrenal medulla PNMT using mixed oligodeoxyribonucleotide probes whose synthesis was based on the partial amino acid sequence of tryptic peptides from the bovine enzyme [Kaneda et al., 1988, (17)]. Using a bovine cDNA as a probe, the authors screened a human pheochromocytoma cDNA library and isolated a cDNA clone with an insert of about 1.0 kb, which contained a complete coding region of the enzyme. Northern blot analysis of human pheochromocytoma polyadenylated RNA using this cDNA insert as the probe demonstrated a single RNA species of about 1,000 nucleotides, suggesting that this clone is a full-length cDNA. The nucleotide sequence showed that human PNMT has 282 amino acid residues with a predicted molecular weight of 30,853, including the initial methionine. The amino acid sequence was 88% homologous to that of bovine enzyme. The PNMT gene was found to consist of 3 exons and 2 introns spanning about 2,100 basepairs. It was demonstrated that in transgenic mice the gene is expressed in adrenal medulla and retina. A hybrid gene consisting of 2 kb of the PNMT 5-prime-flanking region fused to the simian virus 40 early region also resulted in tumor antigen mRNA

expression in adrenal glands and eyes; furthermore, immunocytochemistry showed that the tu antigen was localized in nuclei of adrenal medullary cells and cells of the inner nuclear cell l of the retina, both prominent sites of epinephrine synthesis. The results indicate that enhancer(s) for appropriate expression of the gene in these cell types are in the 2-kb 5-pri
5 flanking region of the gene.

Kaneda et al., 1988 (17), assigned the human PNMT gene to chromosome 17 by Southern analysis of DNA from mouse-human somatic cell hybrids. In 1992 the localization was narro down to 17q21-q22 by linkage analysis using RFLPs related to the PNMT gene and several DNA markers [Hoehe et al., 1992, (18)]. The findings are of interest in light of the description
10 a genetic locus associated with blood pressure regulation in the stroke-prone spontaneo hypertensive rat (SHR-SP) on rat chromosome 10 in a conserved linkage syntenic group co sponding to human chromosome 17q22-q24. See essential hypertension .

MGC9753

This gene maps on chromosome 17, at 17q12 according to RefSeq. It is expressed at very l
15 level. It is defined by cDNA clones and produces, by alternative splicing, 7 different transcr can be obtained (SEQ ID NO:60 to 66 and 83 to 89 ,Table 1), altogether encoding 7 diffe protein isoforms. Of specific interest is the putatively secreted isoform g, encoded by a mRNA, 2.55 kb. Its premessenger covers 16.94 kb on the genome. It has a very long 3' UTR. . The pro (226 aa, MW 24.6 kDa, pI 8.5) contains no Pfam motif. The MGC9753 gene produces,
20 alternative splicing, 7 types of transcripts, predicted to encode 7 distinct proteins. It contains confirmed introns, 10 of which are alternative. Comparison to the genome sequence shows tha introns follow the consensual [gt-ag] rule, 1 is atypical with good support [tg_cg]. The six n abundant isoforms are designated by a) to i) and code for proteins as follows:

- a) This mRNA is 3.03 kb long, its premessenger covers 16.95 kb on the genome. It has a
25 long 3' UTR. The protein (190 aa, MW 21.5 kDa, pI 7.2) contains no Pfam motif. . predicted to localise in the endoplasmic reticulum.
- c) This mRNA is 1.17 kb long, its premessenger covers 16.93 kb on the genome. It may incomplete at the N terminus. The protein (368 aa, MW 41.5 kDa, pI 7.3) contains Pfam motif.
- d) This mRNA is 3.17 kb long, its premessenger covers 16.94 kb on the genome. It has a
30 long 3' UTR and 5'p UTR. . The protein (190 aa, MW 21.5 kDa, pI 7.2) contains no P motif. It is predicted to localise in the endoplasmic reticulum.

- g) This mRNA is 2.55 kb long, its premessenger covers 16.94 kb on the genome. It has a very long 3' UTR. . The protein (226 aa, MW 24.6 kDa, pI 8.5) contains no Pfam motif. It is predicted to be secreted.
- h) This mRNA is 2.68 kb long, its premessenger covers 16.94 kb on the genome. It has a very long 3' UTR. . The protein (320 aa, MW 36.5 kDa, pI 6.8) contains no Pfam motif. It is predicted to localise in the endoplasmic reticulum.
- i) This mRNA is 2.34 kb long, its premessenger covers 16.94 kb on the genome. It may be incomplete at the N terminus. It has a very long 3' UTR. . The protein (217 aa, MW 24.4 kDa, pI 5.9) contains no Pfam motif.
- 10 The MCG9753 gene may be homologue to the CAB2 gene located on chromosome 17q12. The CAB2, a human homologue of the yeast COS16 required for the repair of DNA double-strand breaks was cloned. Autofluorescence analysis of cells transfected with its GFP fusion protein demonstrated that CAB2 translocates into vesicles, suggesting that overexpression of CAB2 may decrease intercellular Mn-
- 15 (2 +) by accumulating it in the vesicles, in the same way as yeast.

Her-2/neu, ERBB2, NGL, TKR1

The oncogene originally called NEU was derived from rat neuro/glioblastoma cell lines. It encodes a tumor antigen, p185, which is serologically related to EGFR, the epidermal growth factor receptor. EGFR maps to chromosome 7. In 1985 it was found, that the human homologue, which they designated NGL (to avoid confusion with neuraminidase, which is also symbolized NEU), maps to 17q12-q22 by in situ hybridization and to 17q21-qter in somatic cell hybrids [Yang-Feng et al., 1985, (19)]. Thus, the SRO is 17q21-q22. Moreover, in 1985 a potential cell surface receptor of the tyrosine kinase gene family was identified and characterized by cloning the gene [Coussens et al., 1985, (20)]. Its primary sequence is very similar to that of the human epidermal growth factor receptor. Because of the seemingly close relationship to the human EGF receptor, the authors called the gene HER2. By Southern blot analysis of somatic cell hybrid DNA and by in situ hybridization, the gene was assigned to 17q21-q22. This chromosomal location of the gene is coincident with the NEU oncogene, which suggests that the 2 genes may in fact be the same; indeed, sequencing indicates that they are identical. In 1988 a correlation between overexpression of NEU protein and the large-cell, comedo growth type of ductal carcinoma was found [van de Vijver et al., 1988, (21)]. The authors found no correlation, however, with lymph-node status or tumor recurrence. The role of HER2/NEU in breast and ovarian cancer was described in 1989,

which together account for one-third of all cancers in women and approximately one-quarter cancer-related deaths in females [Slamon et al., 1989, (22)].

An ERBB-related gene that is distinct from the ERBB gene, called ERBB1 was found in 1986. ERBB2 was not amplified in vulva carcinoma cells with EGFR amplification and did not re-act with EGF receptor mRNA. About 30-fold amplification of ERBB2 was observed in a human adenocarcinoma of the salivary gland. By chromosome sorting combined with velocity sedimentation and Southern hybridization, the ERBB2 gene was assigned to chromosome 17 [Fukushige et al., 1986, (23)]. By hybridization to sorted chromosomes and to metaphase spreads with a genomic probe, they mapped the ERBB2 locus to 17q21. This is the chromosome breakpoint in acute promyelocytic leukemia (APL). Furthermore, they observed amplification and elevated expression of the ERBB2 gene in a gastric cancer cell line. Antibodies against a synthetic peptide corresponding to 14 amino acid residues at the COOH-terminus of a protein deduced from the ERBB2 nucleotide sequence were raised in 1986. With these antibodies, the ERBB2 gene product from adenocarcinoma cells was precipitated and demonstrated to be a 185-kDa glycoprotein with tyrosine kinase activity. A cDNA probe for ERBB2 and by in situ hybridization to APL cells with a 15;17 chromosome translocation located the gene to the proximal side of the breakpoint [Kaneko et al., 1987, (24)]. The authors suggested that both the gene and the breakpoint are located in band 17q21.1 and, further, that the ERBB2 gene is involved in the development of leukemia. In 1987 experiments indicated that NEU and HER2 are both the same as ERBB2 [Di Fiore et al., 1987, (25)]. The authors demonstrated that overexpression alone can convert the gene for a normal growth factor receptor, namely, ERBB2, into an oncogene. The ERBB2 to 17q11-q21 by in situ hybridization [Popescu et al., 1989, (26)]. By in situ hybridization to chromosomes derived from fibroblasts carrying a constitutional translocation between 15 and 17, they showed that the ERBB2 gene was relocated to the derivative chromosome 15; the gene can thus be localized to 17q12-q21.32. By family linkage studies using multiple DNA markers in the 17q12-q21 region the ERBB2 gene was placed on the genetic map of the region.

Interleukin-6 is a cytokine that was initially recognized as a regulator of immune and inflammatory responses, but also regulates the growth of many tumor cells, including prostate cancer. Overexpression of ERBB2 and ERBB3 has been implicated in the neoplastic transformation of prostate cancer. Treatment of a prostate cancer cell line with IL6 induced tyrosine phosphorylation of ERBB2 and ERBB3, but not ERBB1/EGFR. The ERBB2 forms a complex with the gp130 subunit of the IL6 receptor in an IL6-dependent manner. This association was important because the inhibition of ERBB2 activity resulted in abrogation of IL6-induced MAPK activation. Thus, ERBB2 is a critical component of IL6 signaling through the MAP kinase

pathway [Qiu et al., 1998, (27)]. These findings showed how a cytokine receptor can diversify its signaling pathways by engaging with a growth factor receptor kinase.

Overexpression of ERBB2 confers Taxol resistance in breast cancers. Overexpression of ERBB2 inhibits Taxol-induced apoptosis [Yu et al., 1998, (28)]. Taxol activates CDC2 kinase in MDA-MB-435 breast cancer cells, leading to cell cycle arrest at the G2/M phase and, subsequently, apoptosis. A chemical inhibitor of CDC2 and a dominant-negative mutant of CDC2 blocked Taxol-induced apoptosis in these cells. Overexpression of ERBB2 in MDA-MB-435 cells by transfection transcriptionally upregulates CDKN1A which associates with CDC2, inhibits Taxol-mediated CDC2 activation, delays cell entrance to G2/M phase, and thereby inhibits Taxol-induced apoptosis. In CDKN1A antisense-transfected MDA-MB-435 cells or in p21^{-/-} MEF cells, ERBB2 was unable to inhibit Taxol-induced apoptosis. Therefore, CDKN1A participates in the regulation of a G2/M checkpoint that contributes to resistance to Taxol-induced apoptosis in ERBB2-overexpressing breast cancer cells.

A secreted protein of approximately 68 kD was described, designated herstatin, as the product of an alternative ERBB2 transcript that retains intron 8 [Doherty et al., 1999, (29)]. This alternative transcript specifies 340 residues identical to subdomains I and II from the extracellular domain of p185ERBB2, followed by a unique C-terminal sequence of 79 amino acids encoded by intron 8. The recombinant product of the alternative transcript specifically bound to ERBB2-transfected cells and was chemically crosslinked to p185ERBB2, whereas the intron-encoded sequence alone also bound with high affinity to transfected cells and associated with p185 solubilized from cell extracts. The herstatin mRNA was expressed in normal human fetal kidney and liver, but was at reduced levels relative to p185ERBB2 mRNA in carcinoma cells that contained an amplified ERBB2 gene. Herstatin appears to be an inhibitor of p185ERBB2, because it disrupts dimerization, reduces tyrosine phosphorylation of p185, and inhibits the anchorage-independent growth of transformed cells that overexpress ERBB2. The HER2 gene is amplified and HER2 is overexpressed in 25 to 30% of breast cancers, increasing the aggressiveness of the tumor. Finally, it was found that a recombinant monoclonal antibody against HER2 increased the clinical benefit of first-line chemotherapy in metastatic breast cancer that overexpresses HER2 [Slamon et al., 2001, (30)].

30 GRB7

Growth factor receptor tyrosine kinases (GF-RTKs) are involved in activating the cell cycle. Several substrates of GF-RTKs contain Src-homology 2 (SH2) and SH3 domains. SH2 domain-containing proteins are a diverse group of molecules important in tyrosine kinase signaling. Using

the CORT (cloning of receptor targets) method to screen a high expression mouse library, the gene for murine Grb7, which encodes a protein of 535 amino acids, was isolated [Margolis et al., 1992, (31)]. GRB7 is homologous to ras-GAP (ras-GTPase-activating protein). It contains an SH2 domain and is highly expressed in liver and kidney. This gene defines the GRB7 family, whose members include the mouse gene Grb10 and the human gene GRB14.

A putative GRB7 signal transduction molecule and a GRB7V novel splice variant from an invasive human esophageal carcinoma was isolated [Tanaka et al., 1998, (32)]. Although both GRB7 isoforms shared homology with the Mig-10 cell migration gene of *Caenorhabditis elegans*, the GRB7V isoform lacked 88 basepairs in the C terminus; the resultant frameshift led to substitution of an SH2 domain with a short hydrophobic sequence. The wildtype GRB7 protein, but not the GRB7V isoform, was rapidly tyrosyl phosphorylated in response to EGF stimulation in esophageal carcinoma cells. Analysis of human esophageal tumor tissues and regional lymph nodes with metastases revealed that GRB7V was expressed in 40% of GRB7-positive esophageal carcinomas. GRB7V expression was enhanced after metastatic spread to lymph nodes as compared to the original tumor tissues. Transfection of an antisense GRB7 RNA expression construct lowered endogenous GRB7 protein levels and suppressed the invasive phenotype exhibited by esophageal carcinoma cells. These findings suggested that GRB7 isoforms are involved in cell invasion and metastatic progression of human esophageal carcinomas. By sequence analysis, The GRB7 gene was mapped to chromosome 17q21-q22, near the topoisomerase-2 gene [Dong et al., 1997, (33)]. GRB-7 is amplified in concert with HER2 in several breast cancer cell lines and that GRB-7 is overexpressed in both cell lines and breast tumors. GRB-7, through its SH2 domain, binds tightly to HER2 such that a large fraction of the tyrosine phosphorylated HER2 in SKBR-3 cells is bound to GRB-7 [Stein et al., 1994, (34)].

GCSF, CSF3

Granulocyte colony-stimulating factor (or colony stimulating factor-3) specifically stimulates the proliferation and differentiation of the progenitor cells for granulocytes. The partial amino acid sequence of purified GCSF protein was determined, and by using oligonucleotides as probes, several GCSF cDNA clones were isolated from a human squamous carcinoma cell line cDNA library [Nagata et al., 1986, (35)]. Cloning of human GCSF cDNA shows that a single gene codes for a 177- or 180-amino acid mature protein of molecular weight 19,600. The authors found that the GCSF gene has 4 introns and that 2 different polypeptides are synthesized from the same gene by differential splicing of mRNA. The 2 polypeptides differ by the presence or absence of 3 amino acids. Expression studies indicate that both have authentic GCSF activity. A stimulatory activity from a glioblastoma multiform cell line being biologically and biochemically indistinguishable

from GCSF produced by a bladder cell line was found in 1987. By somatic cell hybridization and in situ chromosomal hybridization, the GCSF gene was mapped to 17q11 in the region of the breakpoint in the 15;17 translocation characteristic of acute promyelocytic leukemia [Le Beau et al., 1987, (36)]. Further studies indicated that the gene is proximal to the said breakpoint and that it remains on the rearranged chromosome 17. Southern blot analysis using both conventional and pulsed field gel electrophoresis showed no rearranged restriction fragments. By use of a full-length cDNA clone as a hybridization probe in human-mouse somatic cell hybrids and in flow-sorted human chromosomes, the gene for GCSF was mapped to 17q21-q22 lateron

THRA, THRA1, ERBA, EAR7, ERBA2, ERBA3

Both human and mouse DNA have been demonstrated to have two distantly related classes of ERBA genes and that in the human genome multiple copies of one of the classes exist [Jansson et al., 1983, (37)]. A cDNA was isolated derived from rat brain messenger RNA on the basis of homology to the human thyroid receptor gene [Thompson et al., 1987, (38)]. Expression of this cDNA produced a high-affinity binding protein for thyroid hormones. Messenger RNA from this gene was expressed in tissue-specific fashion, with highest levels in the central nervous system and no expression in the liver. An increasing body of evidence indicated the presence of multiple thyroid hormone receptors. The authors suggested that there may be as many as 5 different but related loci. Many of the clinical and physiologic studies suggested the existence of multiple receptors. For example, patients had been identified with familial thyroid hormone resistance in which peripheral response to thyroid hormones is lost or diminished while neuronal functions are maintained. Thyroidologists recognize a form of cretinism in which the nervous system is severely affected and another form in which the peripheral functions of thyroid hormone are more dramatically affected.

The cDNA encoding a specific form of thyroid hormone receptor expressed in human liver, kidney, placenta, and brain was isolated [Nakai et al., 1988, (39)]. Identical clones were found in human placenta. The cDNA encodes a protein of 490 amino acids and molecular mass of 54,824. Designated thyroid hormone receptor type alpha-2 (THRA2), this protein is represented by mRNAs of different size in liver and kidney, which may represent tissue-specific processing of the primary transcript.

The THRA gene contains 10 exons spanning 27 kb of DNA. The last 2 exons of the gene are alternatively spliced. A 5-kb THRA1 mRNA encodes a predicted 410-amino acid protein; a 2.7-kb THRA2 mRNA encodes a 490-amino acid protein. A third isoform, TR-alpha-3, is derived by alternative splicing. The proximal 39 amino acids of the TH-alpha-2 specific sequences are deleted

in TR-alpha-3. A second gene, THRB on chromosome 3, encodes 2 isoforms of TR-beta 1 alternative splicing. In 1989 the structure and function of the EAR1 and EAR7 genes were elucidated, both located on 17q21 [Miyajima et al., 1989, (40)]. The authors determined that one of the exons in the EAR7 coding sequence overlaps an exon of EAR1, and that the 2 genes are transcribed from opposite DNA strands. In addition, the EAR7 mRNA generates 2 alternative spliced isoforms, referred to as EAR71 and EAR72, of which the EAR71 protein is the human counterpart of the chicken c-erbA protein.

The thyroid hormone receptors, beta, alpha-1, and alpha-2 3 mRNAs are expressed in all tissue examined and the relative amounts of the three mRNAs were roughly parallel. None of the mRNAs was abundant in liver, which is the major thyroid hormone-responsive organ. This led to the assumption that another thyroid hormone receptor may be present in liver. It was found that ERBA, which potentiates ERBB, has an amino acid sequence different from that of other known oncogene products and related to those of the carbonic anhydrases [Debuire et al., 1984, (41)] ERBA potentiates ERBB by blocking differentiation of erythroblasts at an immature stage. Carbonic anhydrases participate in the transport of carbon dioxide in erythrocytes. In 1986 it was shown that the ERBA protein is a high-affinity receptor for thyroid hormone. The cDNA sequence indicates a relationship to steroid-hormone receptors, and binding studies indicate that it is a receptor for thyroid hormones. It is located in the nucleus, where it binds to DNA and activates transcription.

Maternal thyroid hormone is transferred to the fetus early in pregnancy and is postulated to regulate brain development. The ontogeny of TR isoforms and related splice variants in 9 first-trimester fetal brains by semi-quantitative RT-PCR analysis has been investigated. Expression of the TR-beta-1, TR-alpha-1, and TR-alpha-2 isoforms was detected from 8.1 weeks' gestation. An additional truncated species was detected with the TR-alpha-2 primer set, consistent with the TR-alpha-3 splice variant described in the rat. All TR-alpha-derived transcripts were coordinately expressed and increased approximately 8-fold between 8.1 and 13.9 weeks' gestation. A more complex ontogenic pattern was observed for TR-beta-1, suggestive of a nadir between 8.4 and 12.0 weeks' gestation. The authors concluded that these findings point to an important role for the TR-alpha-1 isoform in mediating maternal thyroid hormone action during first-trimester fetal brain development.

The identification of the several types of thyroid hormone receptor may explain the normal variation in thyroid hormone responsiveness of various organs and the selective tissue abnormalities found in the thyroid hormone resistance syndromes. Members of sibships, who were resistant to thyroid hormone action, had retarded growth, congenital deafness, and abnormal

bones, but had normal intellect and sexual maturation, as well as augmented cardiovascular activity. In this family abnormal T3 nuclear receptors in blood cells and fibroblasts have been demonstrated. The availability of cDNAs encoding the various thyroid hormone receptors was considered useful in determining the underlying genetic defect in this family.

5 The ERBA oncogene has been assigned to chromosome 17. The ERBA locus remains on chromosome 17 in the t(15;17) translocation of acute promyelocytic leukemia (APL). The thymidine kinase locus is probably translocated to chromosome 15; study of leukemia with t(17;21) and apparently identical breakpoint showed that TK was on 21q+. By in situ hybridization of a cloned DNA probe of c-erb-A to meiotic pachytene spreads obtained from uncultured
10 spermatocytes it has been concluded that ERBA is situated at 17q21.33-17q22, in the same region as the break that generated the t(15;17) seen in APL. Because most of the grains were seen in 17q22, they suggested that ERBA is probably in the proximal region of 17q22 or at the junction between 17q22 and 17q21.33. By in situ hybridization it has been demonstrated, that that ERBA remains at 17q11-q12 in APL, whereas TP53, at 17q21-q22, is translocated to chromosome 15.
15 Thus, ERBA must be at 17q11.2 just proximal to the breakpoint in the APL translocation and just distal to it in the constitutional translocation.

The aberrant THRA expression in nonfunctioning pituitary tumors has been hypothesized to reflect mutations in the receptor coding and regulatory sequences. They screened THRA mRNA and THRB response elements and ligand-binding domains for sequence anomalies. Screening
20 THRA mRNA from 23 tumors by RNase mismatch and sequencing candidate fragments identified 1 silent and 3 missense mutations, 2 in the common THRA region and 1 that was specific for the alpha-2 isoform. No THRB response element differences were detected in 14 nonfunctioning tumors, and no THRB ligand-binding domain differences were detected in nonfunctioning tumors. Therefore it has been suggested that the novel thyroid receptor mutations
25 may be of functional significance in terms of thyroid receptor action, and further definition of their functional properties may provide insight into the role of thyroid receptors in growth control in pituitary cells.

RAR-alpha

30 A cDNA encoding a protein that binds retinoic acid with high affinity has been cloned [Petkovich et al., 1987, (42)]. The protein was found to be homologous to the receptors for steroid hormones, thyroid hormones, and vitamin D3, and appeared to be a retinoic acid-inducible transacting enhancer factor. Thus, the molecular mechanisms of the effect of vitamin A on embryonic development, differentiation and tumor cell growth may be similar to those described for other

members of this nuclear receptor family. In general, the DNA-binding domain is most highly conserved, both within and between the 2 groups of receptors (steroid and thyroid); Using a cDNA probe, the RAR-alpha gene has been mapped to 17q21 by in situ hybridization [Mattei et al., 1984 (43)]. Evidence has been presented for the existence of 2 retinoic acid receptors, RAR-alpha and RAR-beta, mapping to chromosome 17q21.1 and 3p24, respectively. The alpha and beta forms of RAR were found to be more homologous to the 2 closely related thyroid hormone receptors alpha and beta, located on 17q11.2 and 3p25-p21, respectively, than to any other members of the nuclear receptor family. These observations suggest that the thyroid hormone and retinoic acid receptor evolved by gene, and possibly chromosome, duplications from a common ancestor, which itself diverged rather early in evolution from the common ancestor of the steroid receptor group of the family. They noted that the counterparts of the human RARA and RARB genes are present in both the mouse and chicken. The involvement of RARA at the APL breakpoint may explain why the use of retinoic acid as a therapeutic differentiation agent in the treatment of acute myeloid leukemias is limited to APL. Almost all patients with APL have a chromosomal translocation t(15;17)(q22;q21). Molecular studies reveal that the translocation results in a chimeric gene through fusion between the PML gene on chromosome 15 and the RARA gene on chromosome 17. A hormone-dependent interaction of the nuclear receptors RARA and RXRA with CLOCK and MOP4 has been presented.

CDC18 L, CDC 6

In yeasts, Cdc6 (*Saccharomyces cerevisiae*) and Cdc18 (*Schizosaccharomyces pombe*) associate with the origin recognition complex (ORC) proteins to render cells competent for DNA replication. Thus, Cdc6 has a critical regulatory role in the initiation of DNA replication in yeast. cDNAs encoding *Xenopus* and human homologues of yeast CDC6 have been isolated [Williams et al., 1997, (44)]. They designated the human and *Xenopus* proteins p62(cdc6). Independently, in a yeast 2-hybrid assay using PCNA as bait, cDNAs encoding the human CDC6/Cdc18 homologue have been isolated [Saha et al, 1998, (45)]. These authors reported that the predicted 560-amino acid human protein shares approximately 33% sequence identity with the 2 yeast proteins. On Western blots of HeLa cell extracts, human CDC6/cdc18 migrates as a 66-kD protein. Although Northern blots indicated that CDC6/Cdc18 mRNA levels peak at the onset of S phase and diminish at the onset of mitosis in HeLa cells, the authors found that total CDC6/Cdc18 protein level is unchanged throughout the cell cycle. Immunofluorescent analysis of epitope-tagged protein revealed that human CDC6/Cdc18 is nuclear in G1- and cytoplasmic in S-phase cells, suggesting that DNA replication may be regulated by either the translocation of this protein between the nucleus and cytoplasm or by selective degradation of the protein in the nucleus.

Immunoprecipitation studies showed that human CDC6/Cdc18 associates in vivo with cyclin A, CDK2, and ORC1. The association of cyclin-CDK2 with CDC6/Cdc18 was specifically inhibited by a factor present in mitotic cell extracts. Therefore it has been suggested that if the interaction between CDC6/Cdc18 with the S phase-promoting factor cyclin-CDK2 is essential for the initiation of DNA replication, the mitotic inhibitor of this interaction could prevent a premature interaction until the appropriate time in G1. Cdc6 is expressed selectively in proliferating but not quiescent mammalian cells, both in culture and within tissues in intact animals [Yan et al., 1998, (46)]. During the transition from a growth-arrested to a proliferative state, transcription of mammalian Cdc6 is regulated by E2F proteins, as revealed by a functional analysis of the human Cdc6 promoter and by the ability of exogenously expressed E2F proteins to stimulate the endogenous Cdc6 gene. Immunodepletion of Cdc6 by microinjection of anti-Cdc6 antibody blocked initiation of DNA replication in a human tumor cell line. The authors concluded that expression of human Cdc6 is regulated in response to mitogenic signals through transcriptional control mechanisms involving E2F proteins, and that Cdc6 is required for initiation of DNA replication in mammalian cells.

Using a yeast 2-hybrid system, co-purification of recombinant proteins, and immunoprecipitation, it has been demonstrated later on that an N-terminal segment of CDC6 binds specifically to PR48, a regulatory subunit of protein phosphatase 2A (PP2A). The authors hypothesized that dephosphorylation of CDC6 by PP2A, mediated by a specific interaction with PR48 or a related B-double prime protein, is a regulatory event controlling initiation of DNA replication in mammalian cells. By analysis of somatic cell hybrids and by fluorescence in situ hybridization the human p62(cdc6) gene has been mapped to 17q21.3.

TOP2A, TOP2, TOP2 alpha

DNA topoisomerases are enzymes that control and alter the topologic states of DNA in both prokaryotes and eukaryotes. Topoisomerase II from eukaryotic cells catalyzes the relaxation of supercoiled DNA molecules, catenation, decatenation, knotting, and unknotting of circular DNA. It appears likely that the reaction catalyzed by topoisomerase II involves the crossing-over of 2 DNA segments. It has been estimated that there are about 100,000 molecules of topoisomerase II per HeLa cell nucleus, constituting about 0.1% of the nuclear extract. Since several of the abnormal characteristics of ataxia-telangiectasia appear to be due to defects in DNA processing, screening for these enzyme activities in 5 AT cell lines has been performed [Singh et al., 1988, (47)]. In comparison to controls, the level of DNA topoisomerase II, determined by unknotting of P4 phage DNA, was reduced substantially in 4 of these cell lines and to a lesser extent in the fifth.

DNA topoisomerase I, assayed by relaxation of supercoil DNA, was found to be present at normal levels.

The entire coding sequence of the human TOP2 gene has been determined [Tsai-Pflugfelder et al., 1988, (48)].

5 In addition human cDNAs that had been isolated by screening a cDNA library derived from mechlorethamine-resistant Burkitt lymphoma cell line (Raji-HN2) with a *Drosophila* Topo cDNA had been sequenced [Chung et al., 1989, (49)]. The authors identified 2 classes of sequences representing 2 TOP2 isoenzymes, which have been named TOP2A and TOP2B. The sequence of the TOP2A cDNAs is identical to that of an internal fragment of the TOP2 cDNA isolated
10 Tsai-Pflugfelder et al., 1988 (48). Southern blot analysis indicated that the TOP2A and TOP2B cDNAs are derived from distinct genes. Northern blot analysis using a TOP2A-specific probe detected a 6.5-kb transcript in the human cell line U937. Antibodies against a TOP2A peptide recognized a 170-kD protein in U937 cell lysates. Therefore it was concluded that their results provide genetic and immunochemical evidence for 2 TOP2 isozymes. The complete structure of the TOP2A and TOP2B genes has been reported [Lang et al., 1998, (50)]. The TOP2A gene spans approximately 30 kb and contains 35 exons.

Tsai-Pflugfelder et al., 1988 (48) showed that the human enzyme is encoded by a single-copy gene which they mapped to 17q21-q22 by a combination of in situ hybridization of a cloned fragment to metaphase chromosomes and by Southern hybridization analysis with a panel of mouse-human
20 hybrid cell lines. The assignment to chromosome 17 has been confirmed by the study of somatic cell hybrids. Because of co-amplification in an adenocarcinoma cell line, it was concluded that the TOP2A and ERBB2 genes may be closely linked on chromosome 17 [Keith et al., 1992, (51)]. Using probes that detected RFLPs at both the TOP2A and TOP2B loci, the demonstration of heterozygosity at a frequency of 0.17 and 0.37 for the alpha and beta loci, respectively. The mouse homologue was mapped to chromosome 11 [Kingsmore et al., 1993, (52)]. The structure and function of type II DNA topoisomerases has been reviewed [Watt et al., 1994, (53)]. DNA topoisomerase II-alpha is associated with the pol II holoenzyme and is a required component for chromatin-dependent co-activation. Specific inhibitors of topoisomerase II blocked transcription on chromatin templates, but did not affect transcription on naked templates. Addition of purified
25 topoisomerase II-alpha reconstituted chromatin-dependent activation activity in reactions with core pol II. Therefore the transcription on chromatin templates seems to result in the accumulation of superhelical tension, making the relaxation activity of topoisomerase II essential for productive RNA synthesis on nucleosomal DNA.

IGFBP4

Six structurally distinct insulin-like growth factor binding proteins have been isolated and their cDNAs cloned: IGFBP1, IGFBP2, IGFBP3, IGFBP4, IGFBP5 and IGFBP6. The proteins display strong sequence homologies, suggesting that they are encoded by a closely related family of genes.

5 The IGFBPs contain 3 structurally distinct domains each comprising approximately one-third of the molecule. The N-terminal domain 1 and the C-terminal domain 3 of the 6 human IGFBPs show moderate to high levels of sequence identity including 12 and 6 invariant cysteine residues in domains 1 and 3, respectively (IGFBP6 contains 10 cysteine residues in domain 1), and are thought to be the IGF binding domains. Domain 2 is defined primarily by a lack of sequence
10 identity among the 6 IGFBPs and by a lack of cysteine residues, though it does contain 2 cysteines in IGFBP4. Domain 3 is homologous to the thyroglobulin type I repeat unit. Recombinant human insulin-like growth factor binding proteins 4, 5, and 6 have been characterized by their expression in yeast as fusion proteins with ubiquitin [Kiefer et al., 1992, (54)]. Results of the study suggested to the authors that the primary effect of the 3 proteins is the attenuation of IGF activity and
15 suggested that they contribute to the control of IGF-mediated cell growth and metabolism. Moreover, IGFBPs have influence on EGFR and Her-2/neu mediated signaling. Addition of IGFBPs to Her-2/neu overexpressing cells at least in part blocks growth and survival characteristics of the respective cells.

Based on peptide sequences of a purified insulin-like growth factor-binding protein (IGFBP) rat
20 IGFBP4 has been cloned by using PCR [Shimasaki et al., 1990, (55)]. They used the rat cDNA to clone the human ortholog from a liver cDNA library. Human IGFBP4 encodes a 258-amino acid polypeptide, which includes a 21-amino acid signal sequence. The protein is very hydrophilic, which may facilitate its ability as a carrier protein for the IGFs in blood. Northern blot analysis of rat tissues revealed expression in all tissues examined, with highest expression in liver. It was
25 stated that IGFBP4 acts as an inhibitor of IGF-induced bone cell proliferation. The genomic region containing the IGFBP gene. The gene consists of 4 exons spanning approximately 15 kb of genomic DNA has been examined [Zazzi et al., 1998, (56)]. The upstream region of the gene contains a TATA box and a cAMP-responsive promoter.

By in situ hybridization, the IGFBP4 gene was mapped to 17q12-q21 [Bajalica et al., 1992, (57)].
30 Because the hereditary breast-ovarian cancer gene BRCA1 had been mapped to the same region, it has been investigated whether IGFBP4 is a candidate gene by linkage analysis of 22 BRCA1 families; the finding of genetic recombination suggested that it is not the BRCA1 gene [Tonin et al., 1993, (58)].

EBI1, CCR7, CMKBR7

Using PCR with degenerate oligonucleotides, a lymphoid-specific member of the G protein coupled receptor family has been identified and mapped to 17q12-q21.2 by analysis of human/mouse somatic cell hybrid DNAs and fluorescence in situ hybridization. It has been shown that this receptor had been independently identified as the Epstein-Barr-induced cDNA (symbol EBI1) [Birkenbach et al., 1993, (59)]. EBI1 is expressed in normal lymphoid tissues and in several B- and T-lymphocyte cell lines. While the function and the ligand for EBI1 remains unknown, its sequence and gene structure suggest that it is related to receptors that recognize chemoattractants such as interleukin-8, RANTES, C5a, and fMet-Leu-Phe. Like the chemoattractant receptors, EBI1 contains intervening sequences near its 5-prime end; however, EBI1 is unique in that both of its introns interrupt the coding region of the first extracellular domain. Mouse Ebi1 cDNA has been isolated and found to encode a protein with 86% identity to the human homologue.

Subsets of murine CD4⁺ T cells localize to different areas of the spleen after adoptive transfer. Naive and T helper-1 (TH1) cells, which express CCR7, home to the periarteriolar lymphoid sheath, whereas activated TH2 cells, which lack CCR7, form rings at the periphery of the T-cell zones near B-cell follicles. It has been found that retroviral transduction of TH2 cells with CCR7 forced them to localize in a TH1-like pattern and inhibited their participation in B-cell help in vivo but not in vitro. Apparently differential expression of chemokine receptors results in unique cellular migration patterns that are important for effective immune responses.

CCR7 expression divides human memory T cells into 2 functionally distinct subsets. CCR7⁺ memory cells express receptors for migration to inflamed tissues and display immediate effector function. In contrast, CCR7⁻ memory cells express lymph node homing receptors and lack immediate effector function, but efficiently stimulate dendritic cells and differentiate into CCR7⁺ effector cells upon secondary stimulation. The CCR7⁺ and CCR7⁻ T cells, named central memory (T-CM) and effector memory (T-EM), differentiate in a step-wise fashion from naive T cells, persist for years after immunization, and allow a division of labor in the memory response.

CCR7 expression in memory CD8⁺ T lymphocyte responses to HIV and to cytomegalovirus (CMV) tetramers has been evaluated. Most memory T lymphocytes express CD45RO, but a fraction express instead the CD45RA marker. Flow cytometric analyses of marker expression and cell division identified 4 subsets of HIV- and CMV-specific CD8⁺ T cells, representing a lineage differentiation pattern: CD45RA⁺CCR7⁺ (double-positive); CD45RA⁻CCR7⁺; CD45RA⁻CCR7⁻ (double-negative); CD45RA⁺CCR7⁻. The capacity for cell division, as measured by 5-(and 6-)carboxyl-fluorescein diacetate, succinimidyl ester, and intracellular staining for the Ki67

nuclear antigen, is largely confined to the CCR7⁺ subsets and occurred more rapidly in cells that are also CD45RA⁺. Although the double-negative cells did not divide or expand after stimulation, they did revert to positivity for either CD45RA or CCR7 or both. The CD45RA⁺CCR7⁺ cells, considered to be terminally differentiated, fail to divide, but do produce interferon-gamma and express high levels of perforin. The representation of subsets specific for CMV and for HIV is distinct. Approximately 70% of HIV-specific CD8⁺ memory T cells are double-negative or preterminally differentiated compared to 40% of CMV-specific cells. Approximately 50% of the CMV-specific CD8⁺ memory T cells are terminally differentiated compared to fewer than 10% of the HIV-specific cells. It has been proposed that terminally differentiated CMV-specific cells are poised to rapidly intervene, while double-positive precursor cells remain for expansion and replenishment of the effector cell pool. Furthermore, high-dose antigen tolerance and the depletion of HIV-specific CD4⁺ helper T-cell activity may keep the HIV-specific memory CD8⁺ T cells at the double-negative stage, unable to differentiate to the terminal effector state. B lymphocytes recirculate between B cell-rich compartments (follicles or B zones) in secondary lymphoid organs, surveying for antigen. After antigen binding, B cells move to the boundary of B and T zones to interact with T-helper cells. Furthermore it has been demonstrated that antigen-engaged B cells have increased expression of CCR7, the receptor for the T-zone chemokines CCL19 (also known as ELC) and CCL21, and that they exhibit increased responsiveness to both chemoattractants. In mice lacking lymphoid CCL19 and CCL21 chemokines, or with B cells that lack CCR7, antigen engagement fails to cause movement to the T zone. Using retroviral-mediated gene transfer, the authors demonstrated that increased expression of CCR7 is sufficient to direct B cells to the T zone. Reciprocally, overexpression of CXCR5, the receptor for the B-zone chemokine CXCL13, is sufficient to overcome antigen-induced B-cell movement to the T zone. This points toward a mechanism of B-cell relocalization in response to antigen, and established that cell position *in vivo* can be determined by the balance of responsiveness to chemoattractants made in separate but adjacent zones.

BAF57, SMARCE 1

The SWI/SNF complex in *S. cerevisiae* and *Drosophila* is thought to facilitate transcriptional activation of specific genes by antagonizing chromatin-mediated transcriptional repression. The complex contains an ATP-dependent nucleosome disruption activity that can lead to enhanced binding of transcription factors. The BRG1/brm-associated factors, or BAF, complex in mammals is functionally related to SWI/SNF and consists of 9 to 12 subunits, some of which are homologous to SWI/SNF subunits. A 57-kD BAF subunit, BAF57, is present in higher eukaryotes, but not in yeast. Partial coding sequence has been obtained from purified BAF57 from extracts of

a human cell line [Wang et al., 1998, (60)]. Based on the peptide sequences, they identified cDNAs encoding BAF57. The predicted 411-amino acid protein contains an HMG domain adjacent to a kinesin-like region. Both recombinant BAF57 and the whole BAF complex bind 4-way junction (4WJ) DNA, which is thought to mimic the topology of DNA as it enters or exits the nucleosome. The BAF57 DNA-binding activity has characteristics similar to those of other HMG proteins. It was found that complexes with mutations in the BAF57 HMG domain retain their DNA-binding and nucleosome-disruption activities. They suggested that the mechanism by which mammalian SWI/SNF-like complexes interact with chromatin may involve recognition of higher-order chromatin structure by 2 or more DNA-binding domains. RNase protection studies and Western blot analysis revealed that BAF57 is expressed ubiquitously. Several lines of evidence point toward the involvement of SWI/SNF factors in cancer development [Klochender-Yeivin et al., 2002, (61)]. Moreover, SWI/SNF related genes are assigned to chromosomal regions that are frequently involved in somatic rearrangements in human cancers [Ring et al., 1998, (62)]. In this respect it is interesting that some of the SWI/SNF family members (i.e. SMARCC1, SMARCC2, SMARCD1 and SMARCD22 are neighboring 3 of the eucaryotic ARCHEONs we have identified (i.e. 3p21-p24, 12q13-q14 and 17q respectively) and which are part of the present invention. In this invention we could also map SMARCE1/BAF57 to the 17q12 region by PCR karyotyping.

KRT 10, K10

Keratin 10 is an intermediate filament (IF) chain which belongs to the acidic type I family and is expressed in terminally differentiated epidermal cells. Epithelial cells almost always co-express pairs of type I and type II keratins, and the pairs that are co-expressed are highly characteristic of a given epithelial tissue. For example, in human epidermis, 3 different pairs of keratins are expressed: keratins 5 (type II) and 14 (type I), characteristic of basal or proliferative cells; keratins 1 (type II) and 10 (type I), characteristic of superbasal terminally differentiating cells; and keratins 6 (type II) and 16 (type I) (and keratin 17 [type I]), characteristic of cells induced to hyperproliferate by disease or injury, and epithelial cells grown in cell culture. The nucleotide sequence of a 1,700 bp cDNA encoding human epidermal keratin 10 (56.5 kD) [Darmon et al., 1987, (63)] has been published as well as the complete amino acid sequence of human keratin 10 [Zhou et al., 1988, (64)]. Polymorphism of the KRT10 gene, restricted to insertions and deletions of the glycine-richquasipeptide repeats that form the glycine-loop motif in the C-terminal domain, have been extensively described [Korge et al., 1992, (65)].

By use of specific cDNA clones in conjunction with somatic cell hybrid analysis and in situ hybridization, KRT10 gene has been mapped to 17q12-q21 in a region proximal to the breakpoint at 17q21 that is involved in a t(17;21)(q21;q22) translocation associated with a form of acute

leukemia. KRT10 appeared to be telomeric to 3 other loci that map in the same region: CSF3, ERBA1, and HER2 [Lessin et al., 1988, (66)]. NGFR and HOX2 are distal to K9. It has been demonstrated that the KRT10, KRT13, and KRT15 genes are located in the same large pulsed field gel electrophoresis fragment [Romano et al., 1991, (67)]. A correlation of assignments of the 3 genes makes 17q21-q22 the likely location of the cluster. Transgenic mice expressing a mutant keratin 10 gene have the phenotype of epidermolytic hyperkeratosis, thus suggesting that a genetic basis for the human disorder resides in mutations in genes encoding suprabasal keratins KRT1 or KRT10 [Fuchs et al 1992, (68)]. The authors also showed that stimulation of basal cell proliferation can result from a defect in suprabasal cells and that distortion of nuclear shape or alterations in cytokinesis can occur when an intermediate filament network is perturbed. In a family with keratosis palmaris et plantaris without blistering either spontaneously or in response to mild mechanical or thermal stress and with no involvement of the skin and parts of the body other than the palms and soles, a tight linkage to an insertion-deletion polymorphism in the C-terminal coding region of the KRT10 gene (maximum lod score = 8.36 at theta = 0.00) was found [Rogaev et al., 1993, (69)]. It is noteworthy that it was a rare, high molecular weight allele of the KRT10 polymorphism that segregated with the disorder. The allele was observed once in 96 independent chromosomes from unaffected Caucasians. The KRT10 polymorphism arose from the insertion/deletion of imperfect (CCG)_n repeats within the coding region and gave rise to a variable glycine loop motif in the C-terminal tail of the keratin 10 protein. It is possible that there was a pathogenic role for the expansion of the imperfect trinucleotide repeat.

KRT12, K12

Keratins are a group of water-insoluble proteins that form 10 nm intermediate filaments in epithelial cells. Approximately 30 different keratin molecules have been identified. They can be divided into acidic and basic-neutral subfamilies according to their relative charges, immunoreactivity, and sequence homologies to types I and II wool keratins, respectively. In vivo, a basic keratin usually is co-expressed and 'paired' with a particular acidic keratin to form a heterodimer. The expression of various keratin pairs is tissue specific, differentiation dependent, and developmentally regulated. The presence of specific keratin pairs is essential for the maintenance of the integrity of epithelium. For example, mutations in human K14/K5 pair and the K10/K1 pair underlie the skin diseases, epidermolysis bullosa simplex and epidermolytic hyperkeratosis, respectively. Expression of the K3 and K12 keratin pair have been found in the cornea of a wide number of species, including human, mouse, and chicken, and is regarded as a marker for corneal-type epithelial differentiation. The murine Krt12 (Krt1.12) gene and demonstrated that its expression is corneal epithelial cell specific, differentiation dependent, and

- developmentally regulated [Liu et al., 1993, (70)]. The corneal-specific nature of keratin 12 gene expression signifies keratin 12 plays a unique role in maintaining normal corneal epithelial function. Nevertheless, the exact function of keratin 12 remains unknown and no hereditary human corneal epithelial disorder has been linked directly to the mutation in the keratin 12 gene. As part of a study of the expression profile of human corneal epithelial cells, a cDNA with an open reading frame highly homologous to the cornea-specific mouse keratin 12 gene has been isolated [Nishida et al., 1996, (71)]. To elucidate the function of keratin 12 knockout mice lacking the Krt1.12 gene have been created by gene targeting techniques. The heterozygous mice appear normal. Homozygous mice developed normally and suffered mild corneal epithelial erosion. The corneal epithelia were fragile and could be removed by gentle rubbing of the eyes or brushing. The corneal epithelium of the homozygotes did not express keratin 12 as judged by immunohistochemistry, Western immunoblot analysis with epitope-specific anti-keratin 12 antibodies, Northern hybridization, and in situ hybridization with an antisense keratin 12 riboprobe. The KRT12 gene has been mapped to 17q by study of radiation hybrids and localized to the type I keratin cluster in the interval between D17S800 and D17S930 (17q12-q21) [Nishida et al., 1997, (72)]. The authors presented the exon-intron boundary structure of the KRT12 gene and mapped the gene to 17q12 by fluorescence in situ hybridization. The gene contains 7 introns defining 8 exons that cover the coding sequence. Together the exons and introns span approximately 6 kb of genomic DNA.
- Meesmann corneal dystrophy is an autosomal dominant disorder causing fragility of the anterior corneal epithelium, where the cornea-specific keratins K3 and K12 are expressed. Dominant-negative mutations in these keratins might be the cause of Meesmann corneal dystrophy. Indeed, linkage of the disorder to the K12 locus in Meesmann's original German kindred [Meesmann and Wilke, 1939, (73)] with $Z(\text{max}) = 7.53$ at $\theta = 0.0$ has been found. In 2 pedigrees from Northern Ireland, they found that the disorder co-segregated with K12 in one pedigree and K3 in the other. Heterozygous missense mutations in K3 or in K12 (R135T, V143L,) in each family have been identified. All these mutations occurred in highly conserved keratin helix boundary motifs, where dominant mutations in other keratins have been found to compromise cytoskeletal function severely, leading to keratinocyte fragility.
- The regions of the human KRT12 gene have been sequenced to enable mutation detection for all exons using genomic DNA as a template [Corden et al., 2000, (74)]. The authors found that the human genomic sequence spans 5,919 bp and consists of 8 exons. A microsatellite dinucleotide repeat was identified within intron 3, which was highly polymorphic and which they developed for use in genotype analysis. In addition, 2 mutations in the helix initiation motif of K12 were found

in families with Meesmann corneal dystrophy. In an American kindred, a missense M129T mutation was found in the KRT12 gene. They stated that a total of 8 mutations in the KRT12 gene had been reported.

Genetic interactions within ARCHEONs

- 5 Genes involved in genomic alterations (amplifications, insertions, translocations, deletions, etc.) exhibit changes in their expression pattern. Of particular interest are gene amplifications, which account for gene copy numbers >2 per cell or deletions accounting for gene copy numbers <2 per cell. Gene copy number and gene expression of the respective genes do not necessarily correlate. Transcriptional overexpression needs an intact transcriptional context, as determined by regulatory
- 10 regions at the chromosomal locus (promotor, enhancer and silencer), and sufficient amounts of transcriptional regulators being present in effective combinations. This is especially true for genomic regions, which expression is tightly regulated in specific tissues or during specific developmental stages. ARCHEONs are specified by gene clusters of more than two genes being directly neighbored or in chromosomal order, interspersed by a maximum of 10, preferably 7,
- 15 more preferably 5 or at least 1 gene. The interspersed genes are also co-amplified but do not directly interact with the ARCHEON. Such an ARCHEON may spread over a chromosomal region of a maximum of 20, more preferably 10 or at least 6 Megabases. The nature of an ARCHEON is characterized by the simultaneous amplification and/or deletion and the correlating expression (i.e. upregulation or downregulation respectively) of the encompassed genes in a specific tissue, cell
- 20 type, cellular or developmental state or time point. Such ARCHEONs are commonly conserved during evolution, as they play critical roles during cellular development. In case of these ARCHEONs whole gene clusters are overexpressed upon amplification as they harbor self-regulatory feedback loops, which stabilize gene expression and/or biological effector function even in abnormal biological settings, or are regulated by very similar transcription factor
- 25 combinations, reflecting their simultaneous function in specific tissues at certain developmental stages. Therefore, the gene copy numbers correlates with the expression level especially for genes in gene clusters functioning as ARCHEONs. In case of abnormal gene expressions in neoplastic lesions it is of great importance to know whether the self-regulatory feedback loops have been conserved as they determine the biological activity of the ARCHEON gene members.
- 30 The intensive interaction between genes in ARCHEONs is described for the 17q21 ARCHEON (Fig. 1) by way of illustration not by limitation. In one embodiment the presence or absence of alterations of genes within distinct genomic regions are correlated with each other, as exemplified for breast cancer cell lines (Fig.3 and Fig. 4). This confers to the discovery of the present invention, that multiple interactions of said gene products of defined chromosomal localizations

happen, that according to their respective alterations in abnormal tissue have predictive diagnostic, prognostic and/or preventive and therapeutic value. These interactions are mediated directly or indirectly, due to the fact that the respective genes are part of interconnected or independent signaling networks or regulate cellular behavior (differentiation status, proliferative and/or apoptotic capacity, invasiveness, drug responsiveness, immune modulatory activities) in synergistic, antagonistic or independent fashion. The order of functionally important genes within the ARCHEONs has been conserved during evolution (e.g. the ARCHEON on human chromosome 17q21 is present on mouse chromosome 11). Moreover, it has been found that the 17q21 ARCHEON is also present on human chromosome 3p21 and 12q13, both of which are also involved in amplification events and in tumor development. Most probably these homologous ARCHEONs were formed by duplications and rearrangements during vertebrate evolution. Homologous ARCHEONs consist of homologous genes and/or isoforms of specific gene families (e.g. RARA or RARB or RARG, THRA or THRB, TOP2A or TOP2B, RAB5A or RAB5B, BAF170 or BAF155, BAF60A or BAF60B, WNT5A or WNT5B, IGFBP4 or IGFBP6). Moreover, these regions are flanked by homologous chromosomal gene clusters (e.g. CACN, SCYA, HOX, Keratins). These ARCHEONs have diverged during evolution to fulfill their respective functions in distinct tissues (e.g. the 17q21 ARCHEON has one of its main functions in the central nervous system). Due to their tissue specific function extensive regulatory loops control the expression of the members of each ARCHEON. During tumor development these regulations become critical for the characteristics of the abnormal tissues with respect to differentiation, proliferation, drug responsiveness, invasiveness. It has been found that the co-amplification of genes within ARCHEONs can lead to co-expression of the respective gene products. Some of said genes also exhibit additional mutations or specific patterns of polymorphisms, which are substantial for the oncogenic capacities of these ARCHEONs. It is one of the critical features of such amplicons, which members of the ARCHEON have been conserved during tumor formation (e.g. during amplification and deletion events), thereby defining these genes as diagnostic marker genes. Moreover, the expression of the certain genes within the ARCHEON can be influenced by other members of the ARCHEON, thereby defining the regulatory and regulated genes as target genes for therapeutic intervention. It was also observed, that the expression of certain members of the ARCHEON is sensitive to drug treatment (e.g. TOPO2 alpha, RARA, THRA, HER-2) which defines these genes as "marker genes". Moreover several other genes are suitable for therapeutic intervention by antibodies (CACNB1, EBI1), ligands (CACNB1) or drugs like e.g. kinase inhibitors (CrkRS, CDC6). The following examples of interactions between members of ARCHEONs are offered by way of illustration, not by way of limitation.

EBI1/CCR7 is lymphoid-specific member of the G protein-coupled receptor family. EBI1 recognizes chemoattractants, such as interleukin-8, SCYAs, Rantes, C5a, and fMet-Leu-Phe. The capacity for cell division is largely confined to the CCR7⁺ subsets in lymphocytes. Double-negative cells did not divide or expand after stimulation. CCR7⁻ cells, considered to be terminally differentiated, fail to divide, but do produce interferon-gamma and express high levels of perforin. EBI1 is induced by viral activities such as the Epstein-Barr-Virus. Therefore, EBI1 is associated with transformation events in lymphocytes. A functional role of EBI1 during tumor formation in non-lymphoid tissues has been investigated in this invention. Interestingly, also ERBA and ERBB, located in the same genomic region, are associated with lymphocyte transformation. Moreover, ligands of the receptor (i.e. SCYA5/Rantes) are in genomic proximity on 17q. Abnormal expression of both of these factors in lymphoid and non-lymphoid tissues establishes an autogulatory feedback loop, inducing signaling events within the respective cells. Expression of lymphoid factors has effect on immune cells and modulates cellular behavior. This is of particular interest with regard to abnormal breast tissue being infiltrated by lymphocytes. In line with this, another immunomodulatory and proliferation factor is located nearby on 17q21. Granulocyte colony-stimulating factor (GCSF3) specifically stimulates the proliferation and differentiation of the progenitor cells for granulocytes. A stimulatory activity from a glioblastoma multiforme cell line being biologically and biochemically indistinguishable from GCSF produced by a bladder cell line has also been found. Colony-stimulating factors not only affects immune cells, but also induce cellular responses of non-immune cells, indicating possible involvement in tumor development upon abnormal expression. In addition several other genes of the 17q21 ARCHEON are involved in proliferation, survival, differentiation of immune cells and/or lymphoblastic leukemia, such as MLLT6, ZNF144 and ZNFN1A3, again demonstrating the related functions of the gene products in interconnected key processes within specific cell types. Aberrant expression of more than one of these genes in non-immune cells constitutes signalling activities, that contribute to the oncogenic activities that derive solely from overexpression of the Her-2/neu gene.

PPARBP has been found in complex with the tumorsuppressor gene of the p53 family. Moreover, PPARBP also binds to PPAR-alpha (PPARA), RAR-alpha (RARA), RXR, THRA and TR-beta-1. Due to it's ability to bind to thyroid hormone receptors it has been named TRIP2 and TRAP220. In this complexes PPARBP affects gene regulatory activities. Interestingly, PPARBP is located in genomic proximity to its interaction partners THRA and RARA. We have found PPARBP to be co-amplified with THRA and RARA in tumor tissue. THRA has been isolated from avian erythroblastosis virus in conjunction with ERBB and therefore was named ERBA. ERBA potentiates ERBB by blocking differentiation of erythroblasts at an immature stage. ERBA has been shown to influence ERBB expression. In this setting deletions of C-terminal portions of the

THRA gene product are of influence. Aberrant THRA expression has also been found nonfunctioning pituitary tumors, which has been hypothesized to reflect mutations in the receptor coding and regulatory sequences. THRA function promotes tumor cell development by regulating gene expression of regulatory genes and by influencing metabolic activities (e.g. of key enzymes of alternative metabolic pathways in tumors such as malic enzyme and genes responsible for lipogenesis). The observed activities of nuclear receptors not only reflect their transactivation potential, but are also due to posttranscriptional activities in the absence or presence of ligand. Co-amplification of THRA /ERBB and ERBB has been shown, but its influence on tumor development has been doubted as no overexpression could be demonstrated in breast tumors [de Vijver et al., 1987, (75)]. THRA and RARA are part of nuclear receptor family whose function can be mediated as monomers, homodimers or heterodimers. RARA regulates differentiation of a broad spectrum of cells. Interactions of hormones with ERBB expression has been investigated. Ligands of RARA can inhibit the expression of amplified ERBB genes in breast tumors [Offerdinger et al., 1998, (76)]. As being part of this invention co-amplification and co-expression of THRA and RARA could be shown. It was also found that multiple genes, which are regulated members of the thyroid hormone receptor - and retinoic acid receptor family, are differentially expressed in tumor samples, corresponding to their genomic alterations (amplification, mutation, deletion). These hormone receptor genes and respective target genes are useful to discriminate patient samples with respect to clinical features.

By expression analysis of multiple normal tissues, tumor samples and tumor cell lines and subsequent clustering of the 17q21 region, it was found that the expression profile of Her-2/neu in positive tumor cells and tumor samples exhibits similarities with the expression pattern of tissues from the central nervous system (Fig. 2). This is in line with the observed malformations in the central nervous system of Her-2/neu and THRA knock-out mice. Moreover, it was found that NEUROD2, a nuclear factor involved specifically in neurogenesis, is commonly expressed in the respective samples. This led to the definition of the 17q21 Locus as being an "ARCHAEO" whose primary function in normal organ development is defined to the central nervous system. Surprisingly, the expression of NEUROD2 was affected by therapeutic intervention. Strikingly also ZNF144, TEM7, PIP5K and PPP1R1B are expressed in neuronal cells, where they display diverse tissue specific functions.

In addition Her-2/neu is often co-amplified with GRB7, a downstream member of the signaling cascade being involved in invasive properties of tumors. Surprisingly, we have found another member of the Her-2/neu signaling cascade being overexpressed in primary breast tumors TOB1 (= "Transducer of ERBB signaling"). Strong overexpression of TOB1 correlated with weak

overexpression of Her-2/neu, already indicating its involvement in oncogenic signaling activities. Amplification of Her-2/neu has been assigned to enhanced proliferative capacity, due to the identified downstream components of the signaling cascade (e.g. Ras-Raf-MAPK). In this respect it was surprising that some cdc genes, which are cell cycle dependent kinases, are part of the amplicons, which upon altered expression have great impact on cell cycle progression.

The ARCHEONS on 17q21 and 12q13 are very closely related, as they do not only harbor isoforms of specific genes (e.g. CACNB1 vs. CACNB3, ERBB2 vs. ERBB3, RARA vs. RARG, see below), but are even flanked by whole gene clusters, consisting of multiple isoforms of one gene family positioned in tandem, such as the keratin and the HOX gene cluster. In this respect the simultaneous presence of keratins and receptors of the EGFR family, i.e. ERBB2 (Her-2/neu) and ERBB3 (Her-3) is of special interest, as the expression of individual keratins is very tightly controlled by the EGFR signalling pathway.

Keratins are a group of water-insoluble proteins that form 10 nm intermediate filaments in epithelial cells. Approximately 30 different keratin molecules have been identified. They can be divided into acidic and basic-neutral subfamilies according to their relative charges, immunoreactivity, and sequence homologies to types I and II wool keratins, respectively. In vivo, a basic keratin usually is co-expressed and 'paired' with a particular acidic keratin to form a heterodimer. The expression of various keratin pairs is tissue specific, differentiation dependent, and developmentally regulated. The presence of specific keratin pairs is essential for the maintenance of the integrity of epithelium. Alterations of keratin expression have been observed in tumor epithelium, with an abnormal keratin pattern being expressed in tumor cells compared to the adjacent normal tissue. Mutations in human K14/K5 pair and the K10/K1 pair underlie skin diseases such as epidermolysis bullosa simplex and epidermolytic hyperkeratosis. The expression of these and other keratins within the skin is tightly regulated. For example, the expression of K14/K5 pair is restricted to the basal cell layer of the skin displaying no overlap with the K10/K1 pair, which is solely expressed in the suprabasal layer. Gene expression is very tightly controlled by an interplay of multiple signalling cascades such as the EGFR, TGFR, sonic hedgehog and wnt-signaling, involving receptor tyrosine kinases and serin threonin kinases. In addition, posttranslational modifications such as serine/threonine and/or tyrosine phosphorylation events affect keratin function, and can be attributed to receptor tyrosine kinase signalling and MAPK and ERK activity. Posttranslational modifications of keratins not only alters the solubility of keratins, but also affects nuclear and signalling functions (e.g. after association with 14-3-3 protein). In addition, we did observe genomic alteration of the keratin gene clusters perturbing keratin expression pattern.

Moreover, the physical interaction of keratins, which are located in ARCHEONs of different chromosomes and whose cell type specific expression at distinct differentiation status is regulated by members of the same ARCHEONs is a superb example of the genetic interaction of ARCHEON genes. Examples of this tight interaction between the 12q13 and 17q21 ARCHEONs are the expression and physical interaction of keratin 5 (basic keratin Type II located on 12q13) and keratin 14 (acidic keratin Type I located on 17q21) in the basal layer of the skin, which is shut off in the suprabasal layer and compensated by the expression and physical interaction of keratin 1 (basic keratin Type II located on 12q13) and keratin 10 (acidic keratin Type I located on 17q21). Diverse control mechanisms confer this exclusive expression control including chromosomal positioning and growth factor signaling activities. Interestingly, critical keratins are chromosomally positioned in an ordered fashion reflecting their related but exclusive function in different keratin pairs and in specific tissues, resembling the structure and function relationship of the hox gene clusters on the same chromosomes. Moreover, keratins whose mutation result in specific skin disorders (e.g. mutation of K5 and K14 results in hand and foot syndrome) are located at similar positions within the ARCHEONs on chromosome 17q21 and 12q13. The genes are in close proximity to genes involved in signaling events (e.g. ERBBs and RARs) regulating proliferation, differentiation and apoptotic events also in the skin tissue. For example Her-2/neu is specifically expressed within the basal layer of the skin, where asymmetric cell divisions of adult stem cells or early progenitor cells thereof give rise to a non-differentiated daughter cell residing in the basal layer and a differentiating daughter cell which is subsequently moving to the suprabasal compartment. These asymmetric cell divisions guarantee the self-renewal and the cellular homeostasis of the skin tissue. This is of importance for the biological functions of the skin such as barrier function towards the environmental stress including infectious agents. Perturbation of the signalling activities within the skin results in diseases similar to the hereditary disorders reflecting mutations of specific keratin genes. In clinical studies it has been shown, that blocking EGFR signalling by antibody-treatment (e.g. cetuximab) and small molecule inhibitors (e.g. Iressa) targeted to the receptor tyrosin kinases can result in skin diseases (e.g. acne-like rash) of grade I, II or III. It is part of this invention, that these skin diseases not only reflect side effects of the respective treatments, but are an example for systemic changes occurring as a consequence of therapeutic regimen, thereby indicating susceptibility of the endogenous signaling network to the therapeutic agents. This observation can have consequences on therapeutic decisions, as the therapeutic regimen are normally stopped or is reduced upon occurrence of side effects. However, as the side effects (e.g. the skin diseases occurring under anti growth factor treatment) are indicative of response to treatment (e.g. tumor shrinkage), the treatment should be endured even though "adverse" drug responses occur and side effects should be treated separately by agents

softening the symptoms. Skin diseases such as rash and hand and foot syndrom are just examples for a given side effect under a given treatment (i.e. anti tumor therapy), that can be used for response correlation.

5 Similarly to blocking receptor molecules itself, blocking downstream members of these signaling cascades results mainly in skin diseases (e.g. hand-and-foot syndroms). Surprisingly, we did observe, that treating tumor cells with agents blocking the EGFR/Her-2/neu signaling (e.g. Cetuximab, Iressa, Herceptin, RAF kinase inhibitor, etc.) shifts the expression of specific keratins being part of the ARCHEONS described in this invention. Moreover, the altered expression of
10 keratins in tumor cells of patients is paralleled by a shift of keratin expression in the keratinocytes of the skin of the very same patient. Perturbation of keratin expression and or post-transcriptional modification in the skin tissue seems to resemble the suscepibility of the endogenous growth factor signaling pathways to the respective treatment. The resulting skin diseases are therefore at least
15 some extent indicative of the tumor responsiveness to the regimen. This endogenous responsiveness to anti growth factor signaling agents can also be delineated from polymorphisms and genetic alterations (e.g. mutations) being present within the ARCHEON described in this invention. Of particular interest are in this context polymorphisms being present in the keratin genes. However, polymorphisms within keratins, keratin related genes and/or genes functionally connected to the keratin-based cytoskeleton, not necessarily being present within the ARCHEONS described, are also of importance according to their physical interaction with the respective gene
20 products (e.g. ITGB4). It is part of this invention, that the responsiveness of a given tumor to anti growth factor treatment relates to the genetic predisposition of the respective signaling pathway members and target genes, which include keratins and related genes, that are markers for proliferation, differentiation and apoptosis in normal tissues, such as skin tissue. This knowledge can be used to predict the responsiveness of a tumor based on the characterization of surrog
25 tissues, such as skin, blood and any other normal tissue containing the above mentioned genes and/or gene products. For example the responsiveness to Iressa, RAF-kinase inhibitor and antibody based therapies targeting EGFR and Her-2/neu can be delineated from punch biopsies of the skin (preferably by comparison of pre- and/or post-treatment samples) or blood samples by determining the expression pattern or genetic characterization of keratin or keratin-related genes of
30 an individual patient. Moreover, the responsiveness of such surrogate tissues can then be correlated to the tumor phenotype and the responsiveness of a tumor to the respective treatment, thereby predict therapy outcome. The examples of surrogate tissues are given by way of illustration and not by limitation.

It is yet another embodiment of the invention, that adverse drug responses such as heart toxicities can also be deduced from characteristics of the ARCHEONs described. Of particular interest are the ARCEONs at 17q12-24, 12q13 and 3q21-26. It is known that anthracyclin based, anti-cancer regimens result in heart toxicities (such as dilated cardiomyopathies), as can be deduced e.g. by LVEF measurements. Moreover, anthracyclin pretreated patients have significantly increased heart toxicity events upon subsequent Herceptin™ based regimen. Interestingly, the ARCHEONs described in this invention not only harbor the primary targets of these therapies (i.e. topoisomerases and Her-2/neu), but also important structural and functional genes (Telethonin, PNMT, CACNB1, PPARBP, Her-2/neu, Her4) for muscle function including heart muscle function. These genes are involved in central processes of heart muscle function, such as tyrosine phosphorylation, serine/threonine phosphorylation, calcium influx, regulating e.g. central structural proteins such as titin. Moreover, these genes can be colocalized in heart muscles, displaying their functional interplay in this tissue. In mouse models, the mislocalization of telethonin and the genetic inactivation of Her-2/neu, Her4 and Neuregulin result in a similar phenotype as can be seen for cancer patients being treated with diverse anti-cancer drugs. The synergistic adverse drug response effect seen for the combinatorial treatment with anthracyclin and Herceptin™. Delineation of polymorphisms and haplotypes of the respective genes, genomic region and/or the ARCHEON structure are indicative of the susceptibility to suffer from heart toxicities upon anti-cancer drug treatment. This is important for therapy decisions and cancer treatment management, as the prior therapies conducted exclude subsequent treatment options. For example, anthracyclin-based pretreatment can exclude subsequent Herceptin™ treatment or lead to reduced dosages, if possible heart toxicities (e.g. dilated cardiomyopathies) cannot be excluded.

According to the observations described above the following examples of genes at 3q21-26 are offered by way of illustration, not by way of limitation.

→ WNT5A, CACNA1D, THRB, RARB, TOP2B, RAB5B, SMARCC1 (BAF155), RAF, WNT7A

The following examples of genes at 12q13 are offered by way of illustration, not by way of limitation.

→ CACNB3, Keratins, ERBB3, NR4A1, RAB5/13, RARG, STAT6, WNT10B, (GCN5), (SAS: Sarcoma Amplified Sequence), SMARCC2 (BAF170), SMARCD1 (BAF60A), (GAS41: Glioma Amplified Sequence), (CHOP), Her3, KRTHB, HOX C, IGFBP6, WNT5B

There is cross-talk between the amplified ARCHEONs described above and some other highly amplified genomic regions locate approximately at 1p13, 1q32, 2p16, 2q21, 3p12, 5p13, 6p12, 7p12, 7q21, 8q23, 11q13, 13q12, 19q13, 20q13 and 21q11. The above mentioned chromosomal regions are described by way of illustration not by way of limitation, as the amplified regions often span larger and/or overlapping positions at these chromosomal positions.

Additional alterations of non-transcribed genes, pseudogenes or intergenic regions of said chromosomal locations can be measured for prediction, diagnosis, prognosis, prevention and treatment of malignant neoplasia and breast cancer in particular. Some of the genes or genomic regions have no direct influence on the members of the ARCHEONs or the genes within distinct chromosomal regions but still retain marker gene function due to their chromosomal positioning in the neighborhood of functionally critical genes (e.g. Telethonin neighboring the Her-2/neu gene).

Clinical Relevance of the genes which are part of the 17q21 Archeon for Response to Herceptin treatment

Clinical Samples of patients being treated with Herceptin, docetaxel, paclitaxel, taxotere, carboplatin, cisplatin, oxaliplatin, vinorelbine, tamoxifen, anastrozole, letrozole, tamoxifen, epirubicin, doxorubicin and CMF were obtained. Primary tumor tissues and lymphnode tissues were obtained from neoadjuvant and adjuvant settings. In addition, biopsy material of first and second line therapies was obtained in some cases from metastatic lesions. These samples included formalin-fixed and paraffin-embedded material or fresh tissue from primary tumours and metastatic lesions of the respective patients. Moreover, whole blood, serum and plasma samples were included in the analysis.

Multiparametric, clinical assessment of the response to Herceptin in combination with chemotherapeutics (e.g. docetaxel, taxotere, paclitaxel, vinorelbine, carboplatin, cisplatin), or other therapies described below, was performed, based on clinical information, such as histological parameters (TNM-Stage, AJCC grade), standard molecular markers (IHC staining for estrogen receptor, progesteron receptor, Her-2/neu) and sonographical or radiological assessment (e.g. CT). In addition to combinatorial treatment, samples from single agent therapies were evaluated. Response to treatment was evaluated according to international standards. The ARCHEON genes were analyzed on DNA, RNA or protein level. Normalization of the ARCHEON genes was done by intra- or extrachromosomal reference genes (see EXAMPLE 3 below) or by housekeeping genes of diverse expression level.

We could delineate specific regions of the ARCHEON to be informative for the response to Herceptin-based therapy. As depicted below, genes that are located towards the centromer telomer of an individual chromosome in relation to a centrally localized gene within ARCHEON (e.g. Her-2/neu in the 17q21 ARCHEON) are in the following named to „centromeric“ and „telomeric“, respectively. Of particular interest for response to Herceptin-based treatment are genes being centromeric from the Her-2/neu gene locus on 17q21. The integrity this centromeric ARCHEON region is of importance for the phenotype of Her-2/neu positive tumors. Genetic alteration in the chromosomal region of PIP5K2B, FLJ20291, MLN50, TEM CACNB1, RPL19, MGC15482, PPARBP, CrkRS are critical for clinical outcome of Her-2/neu positive breast tumors. Of particular interest is the centromeric breakpoint region of the 17q2 ARCHEON nearby the genes TEM7, CACNB1, CrkRS and PPARBP. Her-2/neu positive tumors bearing elevated gene copy numbers of TEM7, CACNB1, CrkRS and PPARBP compared to other Her-2/neu positive tumors and/or normal tissue controls do have a worsened clinical outcome and a poor response to Herceptin based treatment. The genes within this region are involved in calcium and inositol signalling, which is fundamental with regard to cell survival mechanisms (e.g. CACNB1, PPP1R1B and PIP5K2B). Overexpression of CrkRS is of importance for the tumor phenotype, as its kinase activity regulates the RNA polymerase II holoenzyme complex. Especially the phosphorylation of the C-terminal domain and its associated components not only has influence on the general activity of the enzyme complex, but also affects gene products, whose importance for tumor cell growth has been demonstrated and some of which are part of the ARCHEONS (e.g. the SWI/SNF components SMARCs, e.g. SMARCC2, are critical for RB mediated tumor suppression). Phosphorylation of SMARCs is tightly regulated during cell cycle progression and affects the biological function of the SMARCs (influence on activity, stability and cellular localization). Altered phosphorylation of the RNA polymerase holoenzyme complex by CrkRS therefore most probably affects cell cycle progression. Moreover, the abnormal expression of TEM7, which we found to be elevated in a subclass of Her-2/neu positive tumors, whereas it was originally identified to be a tumor endothelial marker (TEM; see above), points towards an intense interplay between tumor and endothelial cells resulting in a more aggressive behaviour of the respective tumor cells during metastatic processes such as intra- and extravasation. Strikingly, the genes within this region, i.e. ZNF144, TEM7, PIP5K, PPP1R1B and CACNB1, all do have physiological functions within the central nervous system. Therefore, we do assume, that a „neuronal environment“ would be favourable for tumor cells overexpressing these genes resulting in growth and survival advantages for these particular tumor cells. In accordance with this, it is observed that Herceptin resistant metastasis frequently occur in the brain. So far it has been discussed, that this observation refers to toxicological problems such as drug-

bioavailability with respect to the blood brain barrier. It is part of this invention, that genes which are normally expressed within neuronal cells are integral part of the centromeric gene cluster of the ARCHEON on chromosome 17q21 and are involved in de novo and acquired resistance to Herceptin based treatment. Independent amplification units and/or deletion of singular genes of this centromeric cluster due to chromosomal breakage interferes with the survival and resistance function of this genomic region. Therefore the continuity of amplification units is another important feature with regard to responsiveness or unresponsiveness to therapy. It is noteworthy to mention, that not only the presence of particular genes, but also the presence of regulatory elements within this genomic region contribute to the above mentioned biological phenotype. Therefore also the loss or gain of regulatory elements within the centromeric part of the ARCHEON is of importance for resistance to anti cancer treatment and therefore part of this invention.

In addition to the alteration of centromeric ARCHEON region, the total length of the ARCHEON with regard to the telomeric-region and the relative gene copy numbers of the amplified genes are of importance. Particularly the integrity of the genomic region harboring the TOP2alpha gene with the surrounding genes THRA, NR1D1, MLN51, WIRE, HsCDC6, RARA, CTEN, IGFBP4, EBI1 and SMARCE1 is of interest. Her-2/neu positive tumors, that are deleted in at least some of this genes exhibit a worsened response to herceptin-based chemotherapy. This demonstrates, that this region is not only of prognostic value for anthracyclin-based therapy, but also of prognostic value for chemotherapeutic treatment with taxol-related agents and platin salts. The amplification, deletion or silencing of this telomeric region is accompanied with altered sensitivity to the above mentioned chemotherapeutics. This is a general feature of tumors bearing alterations (with regard to gene expression and/or amplification of the 17q21 ARCHEON) and not only true for breast cancer. In line with this, we have analyzed ovarian tumors bearing alterations in the 17q21 ARCHEON and correlated the clinical outcome, that was assessed similarly as depicted above, with regard to a platin salt based therapy. Strikingly, tumors with defined genetic patterns within this telomeric regions did develop resistance to this chemotherapeutic regimen. Detecting solely the coamplification of Her-2/neu and TOP2alpha was not as informative with regard to response prediction as a detailed characterization and subsequent response correlation with the region of the THRA, NR1D1, MLN51, WIRE, HsCDC6 and RARA genes. It is part of this invention, that the proliferation status of tumors is affected by genes within ARCHEON regions. The 17q21 ARCHEON determines to at least some extent the proliferation rate of tumor cells. Interestingly, Her-2/neu positive tumors bearing elevated levels of a more limited number of genes, excluding several genes in the telomeric region (i.e. TOP2alpha, HsCDC6) exhibit a relatively slow growth rate, which diminishes the effect of chemotherapeutic drugs targeting proliferating cells and is one

of the reasons for the resistance of these tumors to said agents. Instead, these tumors have a higher capacity with regard to invasiveness and do have a diminished apoptotic rate, which to some extent refers to the signaling of Her-2/neu via GRB7 and AKT kinase (also affected by inositols and calcium, see above), respectively. Several genes within the telomeric region of the
5 ARCHEONs affect Her-2/neu signalling, such as RARA, THRA, IGFBP4, and alter the respective characteristics of the cells including proliferation status.

The ARCHEONs being part of this invention, are not only important for clinical response of tumors to antibody-based therapies raised against EGFR- and Her-2/neu signaling (e.g. Herceptin, 2C4 or cetuximab regimen) and to chemotherapeutic agents, but also are of importance for diverse
10 strategies of anti hormonal treatment (e.g. Tamoxifen, Raloxifen, anastrozol, letrozol, faslodex). In particular, elevated levels of the PPARBP gene and protein and the integrity of the telomeric hormone receptor region of the 17q21 ARCHEON, bearing THRA, NR1D1 and RARA, or its related regions on the other ARCHEONs are of importance for these therapeutic regimens. In a retrospective, clinical study evaluating the above mentioned clinical parameters for adjuvant
15 treatment of breast cancer with tamoxifen, we did observe, that the overexpression of PPARBP has impact on the overall survival of patients receiving this therapy. Overexpression of PPARBP enables activity of estrogen and progesteron receptors irrespective of a bound ligand. Therefore, the deregulation of the PPARBP results in the activity of these hormone receptors in the absence of the hormones and even in the presence of anti-hormones and thereby circumvents the anti
20 tumor effect of anti hormonal strategies resulting in resistance of PPARBP overexpressing cells. In addition overexpression of hormone receptors other than estrogen receptor in tumor cells affects activity of estrogen or the respective anti-hormones by competition for dimerization partners, such as RXR, or transcriptional activator or repressor genes, such as CBP or NCOR. With regard to tamoxifen treatment this clearly diminishes the effect of the anti-hormone, as the pool of the
25 transcriptional cofactors is reduced for the classical mode of action of tamoxifen within the nucleus..

The invention further relates to the use of:

- A) a polynucleotide comprising at least one of the sequences of SEQ ID NO: 1 to 26 or 53 to 75;
- 30 B) a polynucleotide which hybridizes under stringent conditions to a polynucleotide specified in (a) encoding a polypeptide exhibiting the same biological function as specified for the respective sequence in Table 2 or 3

- C) a polynucleotide the sequence of which deviates from the polynucleotide specified in (a) and (b) due to the generation of the genetic code encoding a polypeptide exhibiting the same biological function as specified for the respective sequence in Table 2 or 3
- 5 D) a polynucleotide which represents a specific fragment, derivative or allelic variation of a polynucleotide sequence specified in (a) to (c)
- E) an antisense molecule targeting specifically one of the polynucleotide sequences specified in (a) to (d);
- F) a purified polypeptide encoded by a polynucleotide sequence specified in (a) to (d)
- 10 G) a purified polypeptide comprising at least one of the sequences of SEQ ID NO: 27 to 52 or 76 to 98;
- H) an antibody capable of binding to one of the polynucleotide specified in (a) to (d) or a polypeptide specified in (f) and (g)
- 15 I) a reagent identified by any of the methods of claim 14 to 16 that modulates the amount or activity of a polynucleotide sequence specified in (a) to (d) or a polypeptide specified in (f) and (g)

In the preparation of a composition for the prevention, prediction, diagnosis, prognosis or a medicament for the treatment of malignant neoplasia and breast cancer in particular.

Polynucleotides

20 A „BREAST CANCER GENE“ polynucleotide can be single- or double-stranded and comprise coding sequence or the complement of a coding sequence for a „BREAST CANCER GENE“ polypeptide. Degenerate nucleotide sequences encoding human „BREAST CANCER GENE“ polypeptides, as well as homologous nucleotide sequences which are at least about 50, 55, 60, 65, 70, preferably about 75, 90, 96, or 98% identical to the nucleotide sequences of SEQ ID NO: 1 to 26 or 53 to 75 also are „BREAST CANCER GENE“ polynucleotides. Percent sequence identity

25 between the sequences of two polynucleotides is determined using computer programs such as ALIGN which employ the FASTA algorithm, using an affine gap search with a gap open penalty of -12 and a gap extension penalty of -2. Complementary DNA (cDNA) molecules, species homologues, and variants of „BREAST CANCER GENE“ polynucleotides which encode biologically active „BREAST CANCER GENE“ polypeptides also are „BREAST CANCER

30 GENE“ polynucleotides.

Preparation of Polynucleotides

A naturally occurring „BREAST CANCER GENE“ polynucleotide can be isolated free of other cellular components such as membrane components, proteins, and lipids. Polynucleotides can be made by a cell and isolated using standard nucleic acid purification techniques, or synthesized using an amplification technique, such as the polymerase chain reaction (PCR), or by using an automatic synthesizer. Methods for isolating polynucleotides are routine and are known in the art. Any such technique for obtaining a polynucleotide can be used to obtain isolated „BREAST CANCER GENE“ polynucleotides. For example, restriction enzymes and probes can be used to isolate polynucleotide fragments which comprises „BREAST CANCER GENE“ nucleotide sequences. Isolated polynucleotides are in preparations which are free or at least 70, 80, or 90% free of other molecules.

„BREAST CANCER GENE“ cDNA molecules can be made with standard molecular biology techniques, using „BREAST CANCER GENE“ mRNA as a template. Any RNA isolation technique which does not select against the isolation of mRNA may be utilized for the purification of such RNA samples. See, for example, Sambrook et al., 1989, (77); and Ausubel, F. M. et al. 1989, (78), both of which are incorporated herein by reference in their entirety. Additionally, large numbers of tissue samples may readily be processed using techniques well known to those of skill in the art, such as, for example, the single-step RNA isolation process of Chomczynski, P. (1989, U.S. Pat. No. 4,843,155), which is incorporated herein by reference in its entirety.

„BREAST CANCER GENE“ cDNA molecules can thereafter be replicated using molecular biology techniques known in the art and disclosed in manuals such as Sambrook et al., 1989, (77). An amplification technique, such as PCR, can be used to obtain additional copies of polynucleotides of the invention, using either human genomic DNA or cDNA as a template.

Alternatively, synthetic chemistry techniques can be used to synthesize „BREAST CANCER GENE“ polynucleotides. The degeneracy of the genetic code allows alternate nucleotide sequences to be synthesized which will encode a „BREAST CANCER GENE“ polypeptide or a biologically active variant thereof.

Identification of differential expression

Transcripts within the collected RNA samples which represent RNA produced by differentially expressed genes may be identified by utilizing a variety of methods which are well known to those of skill in the art. For example, differential screening [Tedder, T. F. et al., 1988, (79)], subtractive hybridization [Hedrick, S. M. et al., 1984, (80); Lee, S. W. et al., 1984, (81)]; and, preferably,

differential display (Liang, P., and Pardee, A. B., 1993, U.S. Pat. No. 5,262,311, which is incorporated herein by reference in its entirety), may be utilized to identify polynucleotide sequences derived from genes that are differentially expressed.

5 Differential screening involves the duplicate screening of a cDNA library in which one copy of the library is screened with a total cell cDNA probe corresponding to the mRNA population of one cell type while a duplicate copy of the cDNA library is screened with a total cDNA probe corresponding to the mRNA population of a second cell type. For example, one cDNA probe may correspond to a total cell cDNA probe of a cell type derived from a control subject, while the second cDNA probe may correspond to a total cell cDNA probe of the same cell type derived from
10 an experimental subject. Those clones which hybridize to one probe but not to the other potentially represent clones derived from genes differentially expressed in the cell type of interest in control versus experimental subjects.

Subtractive hybridization techniques generally involve the isolation of mRNA taken from two different sources, e.g., control and experimental tissue, the hybridization of the mRNA or single-
15 stranded cDNA reverse-transcribed from the isolated mRNA, and the removal of all hybridized, and therefore double-stranded, sequences. The remaining non-hybridized, single-stranded cDNAs, potentially represent clones derived from genes that are differentially expressed in the two mRNA sources. Such single-stranded cDNAs are then used as the starting material for the construction of a library comprising clones derived from differentially expressed genes.

20 The differential display technique describes a procedure, utilizing the well known polymerase chain reaction (PCR; the experimental embodiment set forth in Mullis, K. B., 1987, U.S. Pat. No. 4,683,202) which allows for the identification of sequences derived from genes which are differentially expressed. First, isolated RNA is reverse-transcribed into single-stranded cDNA, utilizing standard techniques which are well known to those of skill in the art. Primers for the
25 reverse transcriptase reaction may include, but are not limited to, oligo dT-containing primers, preferably of the reverse primer type of oligonucleotide described below. Next, this technique uses pairs of PCR primers, as described below, which allow for the amplification of clones representing a random subset of the RNA transcripts present within any given cell. Utilizing different pairs of primers allows each of the mRNA transcripts present in a cell to be amplified. Among such
30 amplified transcripts may be identified those which have been produced from differentially expressed genes.

The reverse oligonucleotide primer of the primer pairs may contain an oligo dT stretch of nucleotides, preferably eleven nucleotides long, at its 5' end, which hybridizes to the poly(A) tail

of mRNA or to the complement of a cDNA reverse transcribed from an mRNA poly(A) tail. Second, in order to increase the specificity of the reverse primer, the primer may contain one or more, preferably two, additional nucleotides at its 3' end. Because, statistically, only a subset of the mRNA derived sequences present in the sample of interest will hybridize to such primers, the additional nucleotides allow the primers to amplify only a subset of the mRNA derived sequence present in the sample of interest. This is preferred in that it allows more accurate and complete visualization and characterization of each of the bands representing amplified sequences.

The forward primer may contain a nucleotide sequence expected, statistically, to have the ability to hybridize to cDNA sequences derived from the tissues of interest. The nucleotide sequence may be an arbitrary one, and the length of the forward oligonucleotide primer may range from about 5 to about 13 nucleotides, with about 10 nucleotides being preferred. Arbitrary primer sequences cause the lengths of the amplified partial cDNAs produced to be variable, thus allowing different clones to be separated by using standard denaturing sequencing gel electrophoresis. PCR reaction conditions should be chosen which optimize amplified product yield and specificity, and, additionally, produce amplified products of lengths which may be resolved utilizing standard gel electrophoresis techniques. Such reaction conditions are well known to those of skill in the art, and important reaction parameters include, for example, length and nucleotide sequence of oligonucleotide primers as discussed above, and annealing and elongation step temperatures and reaction times. The pattern of clones resulting from the reverse transcription and amplification of the mRNA of two different cell types is displayed via sequencing gel electrophoresis and compared. Differences in the two banding patterns indicate potentially differentially expressed genes.

When screening for full-length cDNAs, it is preferable to use libraries that have been size-selected to include larger cDNAs. Randomly-primed libraries are preferable, in that they will contain more sequences which contain the 5' regions of genes. Use of a randomly primed library may be especially preferable for situations in which an oligo d(T) library does not yield a full-length cDNA. Genomic libraries can be useful for extension of sequence into 5' nontranscribed regulatory regions.

Commercially available capillary electrophoresis systems can be used to analyze the size or confirm the nucleotide sequence of PCR or sequencing products. For example, capillary sequencing can employ flowable polymers for electrophoretic separation, four different fluorescent dyes (one for each nucleotide) which are laser activated, and detection of the emitted wavelengths by a charge coupled device camera. Output/light intensity can be converted to electrical signal using appropriate software (e.g. GENOTYPER and Sequence NAVIGATOR,

Perkin Elmer; ABI), and the entire process from loading of samples to computer analysis and electronic data display can be computer controlled. Capillary electrophoresis is especially preferable for the sequencing of small pieces of DNA which might be present in limited amounts in a particular sample.

5 Once potentially differentially expressed gene sequences have been identified via bulk techniques such as, for example, those described above, the differential expression of such putatively differentially expressed genes should be corroborated. Corroboration may be accomplished via, for example, such well known techniques as Northern analysis and/or RT-PCR. Upon corroboration, the differentially expressed genes may be further characterized, and may be identified as target
10 and/or marker genes, as discussed, below.

Also, amplified sequences of differentially expressed genes obtained through, for example, differential display may be used to isolate full length clones of the corresponding gene. The full length coding portion of the gene may readily be isolated, without undue experimentation, by molecular biological techniques well known in the art. For example, the isolated differentially
15 expressed amplified fragment may be labeled and used to screen a cDNA library. Alternatively, the labeled fragment may be used to screen a genomic library.

An analysis of the tissue distribution of the mRNA produced by the identified genes may be conducted, utilizing standard techniques well known to those of skill in the art. Such techniques may include, for example, Northern analyses and RT-PCR. Such analyses provide information as
20 to whether the identified genes are expressed in tissues expected to contribute to breast cancer. Such analyses may also provide quantitative information regarding steady state mRNA regulation, yielding data concerning which of the identified genes exhibits a high level of regulation in, preferably, tissues which may be expected to contribute to breast cancer.

Such analyses may also be performed on an isolated cell population of a particular cell type
25 derived from a given tissue. Additionally, standard in situ hybridization techniques may be utilized to provide information regarding which cells within a given tissue express the identified gene. Such analyses may provide information regarding the biological function of an identified gene relative to breast cancer in instances wherein only a subset of the cells within the tissue is thought to be relevant to breast cancer.

30 Identification of co-amplified genes

Genes involved in genomic alterations (amplifications, insertions, translocations, deletions, etc.) are identified by PCR-based karyotyping in combination with database analysis. Of particular

interest are gene amplifications, which account for gene copy numbers >2 per cell. Gene copy number and gene expression of the respective genes often correlates. Therefore clusters of genes being simultaneously overexpressed due to gene amplifications can be identified by expression analysis via DNA-chip technologies or quantitative RTPCR. For example, the altered expression of genes due to increased or decreased gene copy numbers can be determined by GeneArray™ technologies from Affymetrix or qRT-PCR with the TaqMan or iCycler Systems. Moreover combination of RNA with DNA analytic enables highly parallel and automated characterization of multiple genomic regions of variable length with high resolution in tissue or single cell samples. Furthermore these assays enable the correlation of gene transcription relative to gene copy number of target genes. As there is not necessarily a linear correlation of expression level and gene copy number and as there are synergistic or antagonistic effects in certain gene clusters, the identification on the RNA-level is easier and probably more relevant for the biological outcome of the alterations especially in tumor tissue.

Detection of co-amplified genes in malignant neoplasia

Chromosomal changes are commonly detected by FISH (=Fluorescence-In-Situ-Hybridization) and CGH (=Comparative Genomic Hybridization). For quantification of genomic regions genes or intergenic regions can be used. Such quantification measures the relative abundance of multiple genes with respect to each other (e.g. target gene vs. centromeric region or housekeeping genes). Changes in relative abundance can be detected in paraffin-embedded material even after extraction of RNA or genomic DNA. Measurement of genomic DNA has advantages compared to RNA-analysis due to the stability of DNA, which accounts for the possibility to perform also retrospective studies and offers multiple internal controls (genes not being altered, amplified or deleted) for standardization and exact calculations. Moreover, PCR-analysis of genomic DNA offers the advantage to investigate intergenic, highly variable regions or combinations of SNP's (=Single Nucleotide Polymorphisms), RFLPs, VNTRs and STRs (in general polypomorphic markers). Determination of SNPs or polypomorphic markers within defined genomic regions (e.g. SNP analysis by "Pyrosequencing™") has impact on the phenotype of the genomic alterations. For example it is of advantage to determine combinations of polymorphisms or haplotypes in order to characterize the biological potential of genes being part of amplified alleles. Of particular interest are polypomorphic markers in breakpoint regions, coding regions or regulatory regions of genes or intergenic regions. By determining predictive haplotypes with defined biological or clinical outcome it is possible to establish diagnostic and prognostic assays with non-tumor samples from patients. Depending on whether preferably one allele or both alleles to same extent are amplified (= linear or non-linear amplifications) haplotypes can be determined. Overrepresentation of

specific polymorphic markers combinations in cells or tissues with gene amplifications facilitates haplotype determination, as e.g. combinations of heterozygous polymorphic markers in nucleic acids isolated from normal tissues, body fluids or biological samples of one patient become almost homozygous in neoplastic tissue of the very same patient. This "gain of homozygosity" corresponds to the measurement of altered genomic region due to amplification events and is suitable for identification of "gain of function"- alterations in tumors, which result in e.g. oncogenic or growth promoting activities. In contrast, the detection of "losses of heterozygosity" is used for identification of anti-oncogenes, gate keeper genes or checkpoint genes, that suppress oncogenic activities and negatively regulate cellular growth processes. This intrinsic difference clearly opposes the impact of the respective genomic regions for tumor development and emphasizes the significance of "gain of homozygosity" measurements disclosed in this invention. In addition to the analyses on SNPs, a comparative approach of blood leucocyte DNA and tumor DNA based on VNTR detection can reveal the existence of a formerly described ARCHEON. SNP and VNTR sequences and primer sets most suitable for detection of the ARCHEON at 17q11-21 are disclosed in Table 4 and Table 6. Detection, quantification and sizing of such polymorphic markers can be achieved by methods known to those with skill in the art. In one embodiment of this invention we disclose the comparative measurement of amount and size of any of the disclosed VNTRs (Table 6) by PCR amplification and capillary electrophoresis. PCR can be carried out by standart protocols favorably in a linear amplification range (low cycle number) and detection by CE should be carried out by suppliers protocols (e.g. Agilent). More favorably the detection of the VNTRs disclosed in Table 6 can be carried out in a multiplex fashion, utilizing a variety of labeled primers (e.g. fluoreszent, radioactive, bioactive) and a suitable CE detection system (e.g. ABI 310). However the detection can also be performed on slab gels consisting of highly concentrated agarose or polyacrylamide with a monochromal DNA stain. Enhancement of resolution can be achieved by appropriate primer design and length variation to give best results in multiplex PCR.

It is also of interest to determine covalent modifications of DNA (e.g. methylation) or the associated chromatin (e.g. acetylation or methylation of associated proteins) within the altered genomic regions, that have impact on transcriptional activity of the genes. In general, by measuring multiple, short sequences (60-300 bp) these techniques enable high-resolution analysis of target regions, which cannot be obtained by conventional methods such as FISH analytic (2-100 kb). Moreover the PCR-based DNA analysis techniques offer advantages with regard to sensitivity, specificity, multiplexing, time consumption and low amount of patient material required. These techniques can be optimized by combination with microdissection or macrodissection to obtain purer starting material for analysis.

Extending Polynucleotides

In one embodiment of such a procedure for the identification and cloning of full length gene sequences, RNA may be isolated, following standard procedures, from an appropriate tissue or cellular source. A reverse transcription reaction may then be performed on the RNA using an oligonucleotide primer complementary to the mRNA that corresponds to the amplified fragment for the priming of first strand synthesis. Because the primer is anti-parallel to the mRNA, extension will proceed toward the 5' end of the mRNA. The resulting RNA hybrid may then be "tailed" with guanines using a standard terminal transferase reaction, the hybrid may be digested with RNase H, and second strand synthesis may then be primed with a poly-C primer. Using the two primers, the 5' portion of the gene is amplified using PCR. Sequences obtained may then be isolated and recombined with previously isolated sequences to generate a full-length cDNA of the differentially expressed genes of the invention. For a review of cloning strategies and recombinant DNA techniques, see e.g., Sambrook et al., (77); and Ausubel et al., (78).

Various PCR-based methods can be used to extend the polynucleotide sequences disclosed herein to detect upstream sequences such as promoters and regulatory elements. For example, restriction site PCR uses universal primers to retrieve unknown sequence adjacent to a known locus [Sarkar 1993, (82)]. Genomic DNA is first amplified in the presence of a primer to a linker sequence and a primer specific to the known region. The amplified sequences are then subjected to a second round of PCR with the same linker primer and another specific primer internal to the first one. Products of each round of PCR are transcribed with an appropriate RNA polymerase and sequenced using reverse transcriptase.

Inverse PCR also can be used to amplify or extend sequences using divergent primers based on a known region [Triglia et al., 1988, (83)]. Primers can be designed using commercially available software, such as OLIGO 4.06 Primer Analysis software (National Biosciences Inc., Plymouth, Minn.), to be e.g. 2230 nucleotides in length, to have a GC content of 50% or more, and to anneal to the target sequence at temperatures about 68-72°C. The method uses several restriction enzymes to generate a suitable fragment in the known region of a gene. The fragment is then circularized by intramolecular ligation and used as a PCR template.

Another method which can be used is capture PCR, which involves PCR amplification of DNA fragments adjacent to a known sequence in human and yeast artificial chromosome DNA [Lagerstrom et al., 1991, (84)]. In this method, multiple restriction enzyme digestions and ligations also can be used to place an engineered double-stranded sequence into an unknown fragment of the DNA molecule before performing PCR.

Additionally, PCR, nested primers, and PROMOTERFINDER libraries (CLONTECH, Palo Alto, Calif.) can be used to walk genomic DNA (CLONTECH, Palo Alto, Calif.). This process avoids the need to screen libraries and is useful in finding intron/exon junctions.

5 The sequences of the identified genes may be used, utilizing standard techniques, to place the genes onto genetic maps, e.g., mouse [Copeland & Jenkins, 1991, (85)] and human genetic maps [Cohen, et al., 1993, (86)]. Such mapping information may yield information regarding the genes' importance to human disease by, for example, identifying genes which map near genetic regions to which known genetic breast cancer tendencies map.

Identification of polynucleotide variants and homologues or splice variants

10 Variants and homologues of the „BREAST CANCER GENE“ polynucleotides described above also are „BREAST CANCER GENE“ polynucleotides. Typically, homologous „BREAST CANCER GENE“ polynucleotide sequences can be identified by hybridization of candidate polynucleotides to known „BREAST CANCER GENE“ polynucleotides under stringent conditions, as is known in the art. For example, using the following wash conditions: 2 X SSC
15 (0.3 M NaCl, 0.03 M sodium citrate, pH 7.0), 0.1% SDS, room temperature twice, 30 minutes each; then 2 X SSC, 0.1% SDS, 50 EC once, 30 minutes; then 2 X SSC, room temperature twice, 10 minutes each homologous sequences can be identified which contain at most about 25-30% basepair mismatches. More preferably, homologous polynucleotide strands contain 15-25% basepair mismatches, even more preferably 5-15% basepair mismatches.

20 Species homologues of the „BREAST CANCER GENE“ polynucleotides disclosed herein also can be identified by making suitable probes or primers and screening cDNA expression libraries from other species, such as mice, monkeys, or yeast. Human variants of „BREAST CANCER GENE“ polynucleotides can be identified, for example, by screening human cDNA expression libraries. It is well known that the T_m of a double-stranded DNA decreases by 1-1.5°C with every 1% decrease
25 in homology [Bonner et al., 1973, (87)]. Variants of human „BREAST CANCER GENE“ polynucleotides or „BREAST CANCER GENE“ polynucleotides of other species can therefore be identified by hybridizing a putative homologous „BREAST CANCER GENE“ polynucleotide with a polynucleotide having a nucleotide sequence of one of the sequences of the SEQ ID NO: 1 to 26 or 53 to 75 or the complement thereof to form a test hybrid. The melting temperature of the test
30 hybrid is compared with the melting temperature of a hybrid comprising polynucleotides having perfectly complementary nucleotide sequences, and the number or percent of basepair mismatches within the test hybrid is calculated.

Nucleotide sequences which hybridize to „BREAST CANCER GENE“ polynucleotides or their complements following stringent hybridization and/or wash conditions also are „BREAST CANCER GENE“ polynucleotides. Stringent wash conditions are well known and understood in the art and are disclosed, for example, in Sambrook et al., (77). Typically, for stringent hybridization conditions a combination of temperature and salt concentration should be chosen that is approximately 12-20°C below the calculated T_m of the hybrid under study. The T_m of a hybrid between a „BREAST CANCER GENE“ polynucleotide having a nucleotide sequence of one of the sequences of the SEQ ID NO: 1 to 26 or 53 to 75 or the complement thereof and a polynucleotide sequence which is at least about 50, preferably about 75, 90, 96, or 98% identical to one of those nucleotide sequences can be calculated, for example, using the equation below [Bolton and McCarthy, 1962, (88):

$$T_m = 81.5^{\circ}\text{C} - 16.6(\log_{10}[\text{Na}^+]) + 0.41(\%G + C) - 0.63(\%\text{formamide}) - 600/l,$$

where l = the length of the hybrid in basepairs.

Stringent wash conditions include, for example, 4 X SSC at 65°C, or 50% formamide, 4 X SSC at 28°C, or 0.5 X SSC, 0.1% SDS at 65°C. Highly stringent wash conditions include, for example, 0.2 X SSC at 65°C.

The biological function of the identified genes may be more directly assessed by utilizing relevant in vivo and in vitro systems. In vivo systems may include, but are not limited to, animal systems which naturally exhibit breast cancer predisposition, or ones which have been engineered to exhibit such symptoms, including but not limited to the apoE-deficient malignant neoplasia mouse model [Plump et al., 1992, (89)].

Splice variants derived from the same genomic region, encoded by the same pre mRNA can be identified by hybridization conditions described above for homology search. The specific characteristics of variant proteins encoded by splice variants of the same pre transcript may differ and can also be assayed as disclosed. A „BREAST CANCER GENE“ polynucleotide having a nucleotide sequence of one of the sequences of the SEQ ID NO: 1 to 26 or 53 to 75 or the complement thereof may therefor differ in parts of the entire sequence as presented for SEQ ID NO: 60 and the encoded splice variants SEQ ID NO: 61 to 66. These refer to individual proteins SEQ ID NO: 83 to 89. The prediction of splicing events and the identification of the utilized acceptor and donor sites within the pre mRNA can be computed (e.g. Software Package GRAIL or GenomeSCAN) and verified by PCR method by those with skill in the art.

Antisense oligonucleotides

Antisense oligonucleotides are nucleotide sequences which are complementary to a specific DNA or RNA sequence. Once introduced into a cell, the complementary nucleotides combine with natural sequences produced by the cell to form complexes and block either transcription or translation. Preferably, an antisense oligonucleotide is at least 6 nucleotides in length, but can be at least 7, 8, 10, 12, 15, 20, 25, 30, 35, 40, 45, or 50 or more nucleotides long. Longer sequences also can be used. Antisense oligonucleotide molecules can be provided in a DNA construct and introduced into a cell as described above to decrease the level of „BREAST CANCER GENE“ gene products in the cell.

Antisense oligonucleotides can be deoxyribonucleotides, ribonucleotides, peptide nucleic acids (PNAs; described in U.S. Pat. No. 5,714,331), locked nucleic acids (LNAs; described in WO 99/12826), or a combination of them. Oligonucleotides can be synthesized manually or by an automated synthesizer, by covalently linking the 5' end of one nucleotide with the 3' end of another nucleotide with non-phosphodiester internucleotide linkages such as alkylphosphonates, phosphorothioates, phosphorodithioates, alkylphosphonothioates, alkylphosphonates, phosphoramidates, phosphate esters, carbamates, acetamides, carboxymethyl esters, carbonates, and phosphate triesters [Brown, 1994, (126); Sonveaux, 1994, (127) and Uhlmann et al., 1990, (128)].

Modifications of „BREAST CANCER GENE“ expression can be obtained by designing antisense oligonucleotides which will form duplexes to the control, 5', or regulatory regions of the „BREAST CANCER GENE“. Oligonucleotides derived from the transcription initiation site, e.g., between positions 10 and +10 from the start site, are preferred. Similarly, inhibition can be achieved using "triple helix" base-pairing methodology. Triple helix pairing is useful because it causes inhibition of the ability of the double helix to open sufficiently for the binding of polymerases, transcription factors, or chaperons. Therapeutic advances using triplex DNA have been described in the literature [Gee et al., 1994, (129)]. An antisense oligonucleotide also can be designed to block translation of mRNA by preventing the transcript from binding to ribosomes.

Precise complementarity is not required for successful complex formation between an antisense oligonucleotide and the complementary sequence of a „BREAST CANCER GENE“ polynucleotide. Antisense oligonucleotides which comprise, for example, 2, 3, 4, or 5 or more stretches of contiguous nucleotides which are precisely complementary to a „BREAST CANCER GENE“ polynucleotide, each separated by a stretch of contiguous nucleotides which are not complementary to adjacent „BREAST CANCER GENE“ nucleotides, can provide sufficient

targeting specificity for „BREAST CANCER GENE“ mRNA. Preferably, each stretch complementary contiguous nucleotides is at least 4, 5, 6, 7, or 8 or more nucleotides in length. Non-complementary intervening sequences are preferably 1, 2, 3, or 4 nucleotides in length. One skilled in the art can easily use the calculated melting point of an antisense-sense pair to determine the degree of mismatching which will be tolerated between a particular antisense oligonucleotide and a particular „BREAST CANCER GENE“ polynucleotide sequence.

Antisense oligonucleotides can be modified without affecting their ability to hybridize to „BREAST CANCER GENE“ polynucleotide. These modifications can be internal or at one or both ends of the antisense molecule. For example, internucleoside phosphate linkages can be modified by adding cholesteryl or diamine moieties with varying numbers of carbon residues between the amino groups and terminal ribose. Modified bases and/or sugars, such as arabinoside instead of ribose, or a 3', 5' substituted oligonucleotide in which the 3' hydroxyl group or the phosphate group are substituted, also can be employed in a modified antisense oligonucleotide. These modified oligonucleotides can be prepared by methods well known in the art [Agrawal et al., 1992, (130); Uhlmann et al., 1987, (131) and Uhlmann et al., (128)].

Ribozymes

Ribozymes are RNA molecules with catalytic activity [Cech, 1987, (132); Cech, 1990, (133) and Couture & Stinchcomb, 1996, (134)]. Ribozymes can be used to inhibit gene function by cleaving an RNA sequence, as is known in the art (e.g., Haseloff et al., U.S. Patent 5,641,673). The mechanism of ribozyme action involves sequence-specific hybridization of the ribozyme molecule to complementary target RNA, followed by endonucleolytic cleavage. Examples include engineered hammerhead motif ribozyme molecules that can specifically and efficiently catalyze endonucleolytic cleavage of specific nucleotide sequences.

The transcribed sequence of a „BREAST CANCER GENE“ can be used to generate ribozymes which will specifically bind to mRNA transcribed from a „BREAST CANCER GENE“ genomic locus. Methods of designing and constructing ribozymes which can cleave other RNA molecules in trans in a highly sequence specific manner have been developed and described in the art [Haseloff et al., 1988, (135)]. For example, the cleavage activity of ribozymes can be targeted to specific RNAs by engineering a discrete "hybridization" region into the ribozyme. The hybridization region contains a sequence complementary to the target RNA and thus specifically hybridizes with the target [see, for example, Gerlach et al., EP 0 321201].

Specific ribozyme cleavage sites within a „BREAST CANCER GENE“ RNA target can be identified by scanning the target molecule for ribozyme cleavage sites which include the following sequences: GUA, GUU, and GUC. Once identified, short RNA sequences of between 15 and 20 ribonucleotides corresponding to the region of the target RNA containing the cleavage site can be evaluated for secondary structural features which may render the target inoperable. Suitability of candidate „BREAST CANCER GENE“ RNA targets also can be evaluated by testing accessibility to hybridization with complementary oligonucleotides using ribonuclease protection assays. Longer complementary sequences can be used to increase the affinity of the hybridization sequence for the target. The hybridizing and cleavage regions of the ribozyme can be integrally related such that upon hybridizing to the target RNA through the complementary regions, the catalytic region of the ribozyme can cleave the target.

Ribozymes can be introduced into cells as part of a DNA construct. Mechanical methods, such as microinjection, liposome-mediated transfection, electroporation, or calcium phosphate precipitation, can be used to introduce a ribozyme-containing DNA construct into cells in which it is desired to decrease „BREAST CANCER GENE“ expression. Alternatively, if it is desired that the cells stably retain the DNA construct, the construct can be supplied on a plasmid and maintained as a separate element or integrated into the genome of the cells, as is known in the art. A ribozyme-encoding DNA construct can include transcriptional regulatory elements, such as a promoter element, an enhancer or UAS element, and a transcriptional terminator signal, for controlling transcription of ribozymes in the cells.

As taught in Haseloff et al., U.S Pat. No. 5,641,673, ribozymes can be engineered so that ribozyme expression will occur in response to factors which induce expression of a target gene. Ribozymes also can be engineered to provide an additional level of regulation, so that destruction of mRNA occurs only when both a ribozyme and a target gene are induced in the cells.

Polypeptides

“BREAST CANCER GENE” polypeptides according to the invention comprise an polypeptide selected from SEQ ID NO: 27 to 52 and 76 to 98 or encoded by any of the polynucleotide sequences of the SEQ ID NO: 1 to 26 and 53 to 75 or derivatives, fragments, analogues and homologues thereof. A “BREAST CANCER GENE” polypeptide of the invention therefore can be a portion, a full-length, or a fusion protein comprising all or a portion of a “BREAST CANCER GENE” polypeptide.

Protein Purification

„BREAST CANCER GENE“ polypeptides can be purified from any cell which expresses the enzyme, including host cells which have been transfected with „BREAST CANCER GENE“ expression constructs. Breast tissue is an especially useful source of „BREAST CANCER GENE“ polypeptides. A purified „BREAST CANCER GENE“ polypeptide is separated from other compounds which normally associate with the „BREAST CANCER GENE“ polypeptide in the cell, such as certain proteins, carbohydrates, or lipids, using methods well-known in the art. Such methods include, but are not limited to, size exclusion chromatography, ammonium sulfate fractionation, ion exchange chromatography, affinity chromatography, and preparative gel electrophoresis. A preparation of purified „BREAST CANCER GENE“ polypeptides is at least 80% pure; preferably, the preparations are 90%, 95%, or 99% pure. Purity of the preparations can be assessed by any means known in the art, such as SDS-polyacrylamide gel electrophoresis.

Obtaining Polypeptides

„BREAST CANCER GENE“ polypeptides can be obtained, for example, by purification from human cells, by expression of „BREAST CANCER GENE“ polynucleotides, or by direct chemical synthesis.

Biologically Active Variants

„BREAST CANCER GENE“ polypeptide variants which are biologically active, i.e., retain an „BREAST CANCER GENE“ activity, also are „BREAST CANCER GENE“ polypeptides. Preferably, naturally or non-naturally occurring „BREAST CANCER GENE“ polypeptide variants have amino acid sequences which are at least about 60, 65, or 70, preferably about 75, 80, 85, 90, 92, 94, 96, or 98% identical to any of the amino acid sequences of the polypeptides of SEQ ID NO: 27 to 52 or 76 to 98 or the polypeptides encoded by any of the polynucleotides of SEQ ID NO: 1 to 26 or 53 to 75 or a fragment thereof. Percent identity between a putative „BREAST

CANCER GENE" polypeptide variant and of the polypeptides of SEQ ID NO: 27 to 52 or 76 to 98 or the polypeptides encoded by any of the polynucleotides of SEQ ID NO: 1 to 26 or 53 to 75 or a fragment thereof is determined by conventional methods. [See, for example, Altschul *et al.*, 1986, (90) and Henikoff & Henikoff, 1992, (91)]. Briefly, two amino acid sequences are aligned to
5 optimize the alignment scores using a gap opening penalty of 10, a gap extension penalty of 1, and the "BLOSUM62" scoring matrix of Henikoff & Henikoff, (91).

Those skilled in the art appreciate that there are many established algorithms available to align two amino acid sequences. The "FASTA" similarity search algorithm of Pearson & Lipman is a suitable protein alignment method for examining the level of identity shared by an amino acid
10 sequence disclosed herein and the amino acid sequence of a putative variant [Pearson & Lipman, 1988, (92), and Pearson, 1990, (93)]. Briefly, FASTA first characterizes sequence similarity by identifying regions shared by the query sequence (*e.g.*, SEQ ID NO: 1 to 26 or 53 to 75) and a test
sequence that have either the highest density of identities (if the ktup variable is 1) or pairs of identities (if ktup=2), without considering conservative amino acid substitutions, insertions, or
15 deletions. The ten regions with the highest density of identities are then rescored by comparing the similarity of all paired amino acids using an amino acid substitution matrix, and the ends of the regions are "trimmed" to include only those residues that contribute to the highest score. If there are several regions with scores greater than the "cutoff" value (calculated by a predetermined
formula based upon the length of the sequence the ktup value), then the trimmed initial regions are
20 examined to determine whether the regions can be joined to form an approximate alignment with gaps. Finally, the highest scoring regions of the two amino acid sequences are aligned using a modification of the Needleman-Wunsch-Sellers algorithm [Needleman & Wunsch, 1970, (94), and Sellers, 1974, (95)], which allows for amino acid insertions and deletions. Preferred parameters for
FASTA analysis are: ktup=1, gap opening penalty=10, gap extension penalty=1, and substitution
25 matrix=BLOSUM62. These parameters can be introduced into a FASTA program by modifying the scoring matrix file ("SMATRIX"), as explained in Appendix 2 of Pearson, (93).

FASTA can also be used to determine the sequence identity of nucleic acid molecules using a ratio as disclosed above. For nucleotide sequence comparisons, the ktup value can range between one to six, preferably from three to six, most preferably three, with other parameters set as default.

30 Variations in percent identity can be due, for example, to amino acid substitutions, insertions, or deletions. Amino acid substitutions are defined as one for one amino acid replacements. They are conservative in nature when the substituted amino acid has similar structural and/or chemical properties. Examples of conservative replacements are substitution of a leucine with an isoleucine or valine, an aspartate with a glutamate, or a threonine with a serine.

Amino acid insertions or deletions are changes to or within an amino acid sequence. They typically fall in the range of about 1 to 5 amino acids. Guidance in determining which amino acid residues can be substituted, inserted, or deleted without abolishing biological or immunological activity of a „BREAST CANCER GENE“ polypeptide can be found using computer programs well known in the art, such as DNASTAR software. Whether an amino acid change results in a biologically active „BREAST CANCER GENE“ polypeptide can readily be determined by assaying for „BREAST CANCER GENE“ activity, as described for example, in the specific Examples, below. Larger insertions or deletions can also be caused by alternative splicing. Protein domains can be inserted or deleted without altering the main activity of the protein.

Fusion Proteins

Fusion proteins are useful for generating antibodies against „BREAST CANCER GENE“ polypeptide amino acid sequences and for use in various assay systems. For example, fusion proteins can be used to identify proteins which interact with portions of a „BREAST CANCER GENE“ polypeptide. Protein affinity chromatography or library-based assays for protein-protein interactions, such as the yeast two-hybrid or phage display systems, can be used for this purpose. Such methods are well known in the art and also can be used as drug screens.

A „BREAST CANCER GENE“ polypeptide fusion protein comprises two polypeptide segments fused together by means of a peptide bond. The first polypeptide segment comprises at least 25, 50, 75, 100, 150, 200, 300, 400, 500, 600, 700 or 750 contiguous amino acids of an amino acid sequence encoded by any polynucleotide sequences of the SEQ ID NO: 1 to 26 or 53 to 75 or of a biologically active variant, such as those described above. The first polypeptide segment also can comprise full-length „BREAST CANCER GENE“.

The second polypeptide segment can be a full-length protein or a protein fragment. Proteins commonly used in fusion protein construction include β -galactosidase, β -glucuronidase, green fluorescent protein (GFP), autofluorescent proteins, including blue fluorescent protein (BFP), glutathione-S-transferase (GST), luciferase, horseradish peroxidase (HRP), and chloramphenicol acetyltransferase (CAT). Additionally, epitope tags are used in fusion protein constructions, including histidine (His) tags, FLAG tags, influenza hemagglutinin (HA) tags, Myc tags, VSV-G tags, and thioredoxin (Trx) tags. Other fusion constructions can include maltose binding protein (MBP), S-tag, Lex a DNA binding domain (DBD) fusions, GAL4 DNA binding domain fusions, and herpes simplex virus (HSV) BP16 protein fusions. A fusion protein also can be engineered to contain a cleavage site located between the „BREAST CANCER GENE“ polypeptide-encoding

sequence and the heterologous protein sequence, so that the „BREAST CANCER GENE“ polypeptide can be cleaved and purified away from the heterologous moiety.

A fusion protein can be synthesized chemically, as is known in the art. Preferably, a fusion protein is produced by covalently linking two polypeptide segments or by standard procedures in the art of molecular biology. Recombinant DNA methods can be used to prepare fusion proteins, for example, by making a DNA construct which comprises coding sequences selected from any of the polynucleotide sequences of the SEQ ID NO: 1 to 26 and 53 to 75 in proper reading frame with nucleotides encoding the second polypeptide segment and expressing the DNA construct in a host cell, as is known in the art. Many kits for constructing fusion proteins are available from companies such as Promega Corporation (Madison, WI), Stratagene (La Jolla, CA), CLONTECH (Mountain View, CA), Santa Cruz Biotechnology (Santa Cruz, CA), MBL International Corporation (MIC; Watertown, MA), and Quantum Biotechnologies (Montreal, Canada; 1-888-888-8888 DNA-KITS).

Identification of Species Homologues

Species homologues of human a „BREAST CANCER GENE“ polypeptide can be obtained using „BREAST CANCER GENE“ polypeptide polynucleotides (described below) to make suitable probes or primers for screening cDNA expression libraries from other species, such as mice, monkeys, or yeast, identifying cDNAs which encode homologues of a „BREAST CANCER GENE“ polypeptide, and expressing the cDNAs as is known in the art.

Expression of Polynucleotides

To express a „BREAST CANCER GENE“ polynucleotide, the polynucleotide can be inserted into an expression vector which contains the necessary elements for the transcription and translation of the inserted coding sequence. Methods which are well known to those skilled in the art can be used to construct expression vectors containing sequences encoding „BREAST CANCER GENE“ polypeptides and appropriate transcriptional and translational control elements. These methods include in vitro recombinant DNA techniques, synthetic techniques, and in vivo genetic recombination. Such techniques are described, for example, in Sambrook et al., (77) and in Ausubel et al., (78).

A variety of expression vector/host systems can be utilized to contain and express sequences encoding a „BREAST CANCER GENE“ polypeptide. These include, but are not limited to, microorganisms, such as bacteria transformed with recombinant bacteriophage, plasmid, or cosmid DNA expression vectors; yeast transformed with yeast expression vectors, insect cell systems

infected with virus expression vectors (e.g., baculovirus), plant cell systems transformed with virus expression vectors (e.g., cauliflower mosaic virus, CaMV; tobacco mosaic virus, TMV) with bacterial expression vectors (e.g., Ti or pBR322 plasmids), or animal cell systems.

5 The control elements or regulatory sequences are those regions of the vector enhancers, promoter 5' and 3' untranslated regions which interact with host cellular proteins to carry out transcription and translation. Such elements can vary in their strength and specificity. Depending on the vector system and host utilized, any number of suitable transcription and translation elements, including constitutive and inducible promoters, can be used. For example, when cloning in bacterial systems, inducible promoters such as the hybrid lacZ promoter of the BLUESCRIPT phagemid (Stratagene, LaJolla, Calif.) or pSPORT1 plasmid (Life Technologies) and the like can be used. The baculovirus polyhedrin promoter can be used in insect cells. Promoters or enhancers derived from the genomes of plant cells (e.g., heat shock, RUBISCO, and storage protein genes) or from plant viruses (e.g., viral promoters or leader sequences) can be cloned into the vector. In mammalian cell systems, promoters from mammalian genes or from mammalian viruses are preferable. If it is necessary to generate a cell line that contains multiple copies of a nucleotide sequence encoding a „BREAST CANCER GENE“ polypeptide, vectors based on SV40 or EBV can be used with an appropriate selectable marker.

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Bacterial and Yeast Expression Systems

In bacterial systems, a number of expression vectors can be selected depending upon the use intended for the „BREAST CANCER GENE“ polypeptide. For example, when a large quantity of the „BREAST CANCER GENE“ polypeptide is needed for the induction of antibodies, vectors which direct high level expression of fusion proteins that are readily purified can be used. Such vectors include, but are not limited to, multifunctional *E. coli* cloning and expression vectors such as BLUESCRIPT (Stratagene). In a BLUESCRIPT vector, a sequence encoding the „BREAST CANCER GENE“ polypeptide can be ligated into the vector in frame with sequences for the amino terminal Met and the subsequent 7 residues of β -galactosidase so that a hybrid protein is produced. pIN vectors [Van Heeke & Schuster, (17)] or pGEX vectors (Promega, Madison, Wis.) also can be used to express foreign polypeptides as fusion proteins with glutathione S-transferase (GST). In general, such fusion proteins are soluble and can easily be purified from lysed cells by adsorption to glutathione agarose beads followed by elution in the presence of free glutathione. Proteins made in such systems can be designed to include heparin, thrombin, or factor Xa protease cleavage sites so that the cloned polypeptide of interest can be released from the GST moiety at will.

In the yeast *Saccharomyces cerevisiae*, a number of vectors containing constitutive or inducible promoters such as alpha factor, alcohol oxidase, and PGH can be used. For reviews, see Ausubel et al., (4) and Grant et al., (18).

Plant and Insect Expression Systems

If plant expression vectors are used, the expression of sequences encoding „BREAST CANCER GENE“ polypeptides can be driven by any of a number of promoters. For example, viral promoters such as the 35S and 19S promoters of CaMV can be used alone or in combination with the omega leader sequence from TMV [Takamatsu, 1987, (96)]. Alternatively, plant promoters such as the small subunit of RUBISCO or heat shock promoters can be used [Coruzzi et al., 1984, (97); Broglie et al., 1984, (98); Winter et al., 1991, (99)]. These constructs can be introduced into plant cells by direct DNA transformation or by pathogen-mediated transfection. Such techniques are described in a number of generally available reviews.

An insect system also can be used to express a „BREAST CANCER GENE“ polypeptide. For example, in one such system *Autographa californica* nuclear polyhedrosis virus (AcNPV) is used as a vector to express foreign genes in *Spodoptera frugiperda* cells or in *Trichoplusia* larvae. Sequences encoding „BREAST CANCER GENE“ polypeptides can be cloned into a nonessential

region of the virus, such as the polyhedrin gene, and placed under control of the polyhedrin promoter. Successful insertion of „BREAST CANCER GENE“ polypeptides will render the polyhedrin gene inactive and produce recombinant virus lacking coat protein. The recombinant viruses can then be used to infect *S. frugiperda* cells or *Trichoplusia* larvae in which „BREAST CANCER GENE“ polypeptides can be expressed [Engelhard et al., 1994, (100)].

Mammalian Expression Systems

A number of viral-based expression systems can be used to express „BREAST CANCER GENE“ polypeptides in mammalian host cells. For example, if an adenovirus is used as an expression vector, sequences encoding „BREAST CANCER GENE“ polypeptides can be ligated into an adenovirus transcription/translation complex comprising the late promoter and tripartite leader sequence. Insertion in a nonessential E1 or E3 region of the viral genome can be used to obtain a viable virus which is capable of expressing a „BREAST CANCER GENE“ polypeptide in infected host cells [Logan & Shenk, 1984, (101)]. If desired, transcription enhancers, such as the Rous sarcoma virus (RSV) enhancer, can be used to increase expression in mammalian host cells.

Human artificial chromosomes (HACs) also can be used to deliver larger fragments of DNA than can be contained and expressed in a plasmid. HACs of 6M to 10M are constructed and delivered to cells via conventional delivery methods (e.g., liposomes, polycationic amino polymers, or vesicles).

Specific initiation signals also can be used to achieve more efficient translation of sequences encoding „BREAST CANCER GENE“ polypeptides. Such signals include the ATG initiation codon and adjacent sequences. In cases where sequences encoding a „BREAST CANCER GENE“ polypeptide, its initiation codon, and upstream sequences are inserted into the appropriate expression vector, no additional transcriptional or translational control signals may be needed. However, in cases where only coding sequence, or a fragment thereof, is inserted, exogenous translational control signals (including the ATG initiation codon) should be provided. The initiation codon should be in the correct reading frame to ensure translation of the entire insert. Exogenous translational elements and initiation codons can be of various origins, both natural and synthetic. The efficiency of expression can be enhanced by the inclusion of enhancers which are appropriate for the particular cell system which is used [Scharf et al., 1994, (102)].

Host Cells

A host cell strain can be chosen for its ability to modulate the expression of the inserted sequences or to process the expressed „BREAST CANCER GENE“ polypeptide in the desired fashion. Such

modifications of the polypeptide include, but are not limited to, acetylation, carboxylation, glycosylation, phosphorylation, lipidation, and acylation. Posttranslational processing which cleaves a "prepro" form of the polypeptide also can be used to facilitate correct insertion, folding and/or function. Different host cells which have specific cellular machinery and characteristic mechanisms for Post-translational activities (e.g., CHO, HeLa, MDCK, HEK293, and WI38), are available from the American Type Culture Collection (ATCC; 10801 University Boulevard, Manassas, VA 20110-2209) and can be chosen to ensure the correct modification and processing of the foreign protein.

Stable expression is preferred for long-term, high-yield production of recombinant proteins. For example, cell lines which stably express „BREAST CANCER GENE“ polypeptides can be transformed using expression vectors which can contain viral origins of replication and/or endogenous expression elements and a selectable marker gene on the same or on a separate vector. Following the introduction of the vector, cells can be allowed to grow for 12 days in an enriched medium before they are switched to a selective medium. The purpose of the selectable marker is to confer resistance to selection, and its presence allows growth and recovery of cells which successfully express the introduced „BREAST CANCER GENE“ sequences. Resistant clones of stably transformed cells can be proliferated using tissue culture techniques appropriate to the cell type [Freshney et al., 1986, (103)].

Any number of selection systems can be used to recover transformed cell lines. These include, but are not limited to, the herpes simplex virus thymidine kinase [Wigler et al., 1977, (104)] and adenine phosphoribosyltransferase [Lowy et al., 1980, (105)] genes which can be employed in tk⁻ or aprt⁻ cells, respectively. Also, antimetabolite, antibiotic, or herbicide resistance can be used as the basis for selection. For example, dhfr confers resistance to methotrexate [Wigler et al., 1980, (106)], npt confers resistance to the aminoglycosides, neomycin and G418 [Colbere-Garapin et al., 1981, (107)], and als and pat confer resistance to chlorsulfuron and phosphinotricin acetyltransferase, respectively. Additional selectable genes have been described. For example, trpB allows cells to utilize indole in place of tryptophan, or hisD, which allows cells to utilize histinol in place of histidine [Hartman & Mulligan, 1988, (108)]. Visible markers such as anthocyanins, β -glucuronidase and its substrate GUS, and luciferase and its substrate luciferin, can be used to identify transformants and to quantify the amount of transient or stable protein expression attributable to a specific vector system [Rhodes et al., 1995, (109)].

Detecting Expression and gene product

Although the presence of marker gene expression suggests that the „BREAST CANCER GENE“ polynucleotide is also present, its presence and expression may need to be confirmed. For example, if a sequence encoding a „BREAST CANCER GENE“ polypeptide is inserted within
5 marker gene sequence, transformed cells containing sequences which encode a „BREAST CANCER GENE“ polypeptide can be identified by the absence of marker gene function. Alternatively, a marker gene can be placed in tandem with a sequence encoding a „BREAST CANCER GENE“ polypeptide under the control of a single promoter. Expression of the marker gene in response to induction or selection usually indicates expression of the „BREAST CANCER
10 GENE“ polynucleotide.

Alternatively, host cells which contain a „BREAST CANCER GENE“ polynucleotide and which express a „BREAST CANCER GENE“ polypeptide can be identified by a variety of procedures known to those of skill in the art. These procedures include, but are not limited to, DNA-DNA or
15 DNA-RNA hybridization and protein bioassay or immunoassay techniques which include membrane, solution, or chip-based technologies for the detection and/or quantification of polynucleotide or protein. For example, the presence of a polynucleotide sequence encoding a „BREAST CANCER GENE“ polypeptide can be detected by DNA-DNA or DNA-RNA hybridization or amplification using probes or fragments or fragments of polynucleotides encoding
20 a „BREAST CANCER GENE“ polypeptide. Nucleic acid amplification-based assays involve the use of oligonucleotides selected from sequences encoding a „BREAST CANCER GENE“ polypeptide to detect transformants which contain a „BREAST CANCER GENE“ polynucleotide.

A variety of protocols for detecting and measuring the expression of a „BREAST CANCER GENE“ polypeptide, using either polyclonal or monoclonal antibodies specific for the polypeptide, are known in the art. Examples include enzyme-linked immunosorbent assay (ELISA),
25 radioimmunoassay (RIA), and fluorescence activated cell sorting (FACS). A two-site, monoclonal-based immunoassay using monoclonal antibodies reactive to two non-interfering epitopes on a „BREAST CANCER GENE“ polypeptide can be used, or a competitive binding assay can be employed. These and other assays are described in Hampton et al., (110) and Maddox et al., (111).

30 A wide variety of labels and conjugation techniques are known by those skilled in the art and can be used in various nucleic acid and amino acid assays. Means for producing labeled hybridization or PCR probes for detecting sequences related to polynucleotides encoding „BREAST CANCER GENE“ polypeptides include oligo labeling, nick translation, end-labeling, or PCR amplification

using a labeled nucleotide. Alternatively, sequences encoding a „BREAST CANCER GENE“ polypeptide can be cloned into a vector for the production of an mRNA probe. Such vectors are known in the art, are commercially available, and can be used to synthesize RNA probes in vitro by addition of labeled nucleotides and an appropriate RNA polymerase such as T7, T3, or SP6. These procedures can be conducted using a variety of commercially available kits (Amersham Pharmacia Biotech, Promega, and US Biochemical). Suitable reporter molecules or labels which can be used for ease of detection include radionuclides, enzymes, and fluorescent, chemiluminescent, or chromogenic agents, as well as substrates, cofactors, inhibitors, magnetic particles, and the like.

10 Expression and Purification of Polypeptides

Host cells transformed with nucleotide sequences encoding a „BREAST CANCER GENE“ polypeptide can be cultured under conditions suitable for the expression and recovery of the protein from cell culture. The polypeptide produced by a transformed cell can be secreted or stored intracellular depending on the sequence and/or the vector used. As will be understood by those of skill in the art, expression vectors containing polynucleotides which encode „BREAST CANCER GENE“ polypeptides can be designed to contain signal sequences which direct secretion of soluble „BREAST CANCER GENE“ polypeptides through a prokaryotic or eukaryotic cell membrane or which direct the membrane insertion of membrane-bound „BREAST CANCER GENE“ polypeptide.

20 As discussed above, other constructions can be used to join a sequence encoding a „BREAST CANCER GENE“ polypeptide to a nucleotide sequence encoding a polypeptide domain which will facilitate purification of soluble proteins. Such purification facilitating domains include, but are not limited to, metal chelating peptides such as histidine-tryptophan modules that allow purification on immobilized metals, protein A domains that allow purification on immobilized immunoglobulin, and the domain utilized in the FLAGS extension/affinity purification system (Immunex Corp., Seattle, Wash.). Inclusion of cleavable linker sequences such as those specific for Factor Xa or enterokinase (Invitrogen, San Diego, CA) between the purification domain and the „BREAST CANCER GENE“ polypeptide also can be used to facilitate purification. One such expression vector provides for expression of a fusion protein containing a „BREAST CANCER GENE“ polypeptide and 6 histidine residues preceding a thioredoxin or an enterokinase cleavage site. The histidine residues facilitate purification by IMAC (immobilized metal ion affinity chromatography [Porath et al., 1992, (112)], while the enterokinase cleavage site provides a means for purifying the „BREAST CANCER GENE“ polypeptide from the fusion protein. Vectors which contain fusion proteins are disclosed in Kroll et al., (113).

Chemical Synthesis

Sequences encoding a „BREAST CANCER GENE“ polypeptide can be synthesized, in whole or in part, using chemical methods well known in the art (see Caruthers et al., (114) and Horn et al. (115). Alternatively, a „BREAST CANCER GENE“ polypeptide itself can be produced using
5 chemical methods to synthesize its amino acid sequence, such as by direct peptide synthesis using solid-phase techniques [Merrifield, 1963, (116) and Roberge et al., 1995, (117)]. Protein synthesis can be performed using manual techniques or by automation. Automated synthesis can be achieved, for example, using Applied Biosystems 431A Peptide Synthesizer (Perkin Elmer). Optionally, fragments of „BREAST CANCER GENE“ polypeptides can be separately synthesized
10 and combined using chemical methods to produce a full-length molecule.

The newly synthesized peptide can be substantially purified by preparative high performance liquid chromatography [Creighton, 1983, (118)]. The composition of a synthetic „BREAST CANCER GENE“ polypeptide can be confirmed by amino acid analysis or sequencing (e.g., the Edman degradation procedure; see Creighton, (118). Additionally, any portion of the amino acid
15 sequence of the „BREAST CANCER GENE“ polypeptide can be altered during direct synthesis and/or combined using chemical methods with sequences from other proteins to produce a variant polypeptide or a fusion protein.

Production of Altered Polypeptides

As will be understood by those of skill in the art, it may be advantageous to produce „BREAST
20 CANCER GENE“ polypeptide-encoding nucleotide sequences possessing non-natural occurring codons. For example, codons preferred by a particular prokaryotic or eukaryotic host can be selected to increase the rate of protein expression or to produce an RNA transcript having desirable properties, such as a half-life which is longer than that of a transcript generated from the naturally occurring sequence.

The nucleotide sequences disclosed herein can be engineered using methods generally known in the art to alter „BREAST CANCER GENE“ polypeptide-encoding sequences for a variety of reasons, including but not limited to, alterations which modify the cloning, processing, and/or expression of the polypeptide or mRNA product. DNA shuffling by random fragmentation and PCR re-assembly of gene fragments and synthetic oligonucleotides can be used to engineer the
25 nucleotide sequences. For example, site-directed mutagenesis can be used to insert new restriction sites, alter glycosylation patterns, change codon preference, produce splice variants, introduce mutations, and so forth.
30

Predictive, Diagnostic and Prognostic Assays

5 The present invention provides method for determining whether a subject is at risk for developing malignant neoplasia and breast cancer in particular by detecting one of the disclosed polynucleotide markers comprising any of the polynucleotides sequences of the SEQ ID NO: 2 to 6, 8, 9, 11 to 16, 18, 19 or 21 to 26 or 53 to 75 and/or the polypeptide markers encoded thereby or polypeptide markers comprising any of the polypeptide sequences of the SEQ ID NO: 28 to 32, 34, 35, 37 to 42, 44, 45 or 47 to 52 or 76 to 98 or at least 2 of the disclosed polynucleotides selected from SEQ ID NO: 1 to 26 and 53 to 75 or the at least 2 of the disclosed polypeptides selected from SEQ ID NO: 28 to 32 and 76 to 98 for malignant neoplasia and breast cancer in
10 particular.

In clinical applications, biological samples can be screened for the presence and/or absence of the biomarkers identified herein. Such samples are for example needle biopsy cores, surgical resection samples, or body fluids like serum, thin needle nipple aspirates and urine. For example, these methods include obtaining a biopsy, which is optionally fractionated by cryostat sectioning to
15 enrich diseases cells to about 80% of the total cell population. In certain embodiments, polynucleotides extracted from these samples may be amplified using techniques well known in the art. The expression levels of selected markers detected would be compared with statistically valid groups of diseased and healthy samples.

In one embodiment the diagnostic method comprises determining whether a subject has an
20 abnormal mRNA and/or protein level of the disclosed markers, such as by Northern blot analysis, reverse transcription-polymerase chain reaction (RT-PCR), in situ hybridization, immunoprecipitation, Western blot hybridization, or immunohistochemistry. According to the method, cells are obtained from a subject and the levels of the disclosed biomarkers, protein or mRNA level, is determined and compared to the level of these markers in a healthy subject. An
25 abnormal level of the biomarker polypeptide or mRNA levels is likely to be indicative of malignant neoplasia such as breast cancer.

In another embodiment the diagnostic method comprises determining whether a subject has an abnormal DNA content of said genes or said genomic loci, such as by Southern blot analysis, dot blot analysis, fluorescence or colorimetric In Situ hybridization, comparative genomic
30 hybridization, genotyping by VNTR, STS-PCR or quantitative PCR. In general these assays comprise the usage of probes from representative genomic regions. The probes contain at least parts of said genomic regions or sequences complementary or analogous to said regions. In particular intra- or intergenic regions of said genes or genomic regions. The probes can consist of

nucleotide sequences or sequences of analogous functions (e.g. PNAs, Morpholino oligomers) being able to bind to target regions by hybridization. In general genomic regions being altered in said patient samples are compared with unaffected control samples (normal tissue from the same or different patients, surrounding unaffected tissue, peripheral blood) or with genomic regions of the same sample that don't have said alterations and can therefore serve as internal controls. In a preferred embodiment regions located on the same chromosome are used. Alternatively, gonosomal regions and /or regions with defined varying amount in the sample are used. In one favored embodiment the DNA content, structure, composition or modification is compared that lie within distinct genomic regions. Especially favored are methods that detect the DNA content of said samples, where the amount of target regions are altered by amplification and or deletions. In another embodiment the target regions are analyzed for the presence of polymorphisms (e.g. Single Nucleotide Polymorphisms or mutations) that affect or predispose the cells in said samples with regard to clinical aspects, being of diagnostic, prognostic or therapeutic value. Preferably, the identification of sequence variations is used to define haplotypes that result in characteristic behavior of said samples with said clinical aspects.

The following examples of genes in 17q12-21.2 are offered by way of illustration, not by way of limitation.

One embodiment of the invention is a method for the prediction, diagnosis or prognosis of malignant neoplasia by the detection of at least 10, at least 5, or at least 4, or at least 3 and more preferably at least 2 markers whereby the markers are genes and fragments thereof and/or genomic nucleic acid sequences that are located on one chromosomal region which is altered in malignant neoplasia.

One further embodiment of the invention is method for the prediction, diagnosis or prognosis of malignant neoplasia by the detection of at least 10, at least 5, or at least 4, or at least 3 and more preferably at least 2 markers whereby the markers (a) are genes and fragments thereof and/or genomic nucleic acid sequences that are located on one or more chromosomal region(s) which is/are altered in malignant neoplasia and (b) functionally interact as (i) receptor and ligand or (ii) members of the same signal transduction pathway or (iii) members of synergistic signal transduction pathways or (iv) members of antagonistic signal transduction pathways or (v) transcription factor and transcription factor binding site.

In one embodiment, the method for the prediction, diagnosis or prognosis of malignant neoplasia and breast cancer in particular is done by the detection of:

- 5
- (a) polynucleotide selected from the polynucleotides of the SEQ ID NO: 2 to 6, 8, 9, 11 to 16, 18, 19, 21 to 26 or 53 to 75;
 - (b) a polynucleotide which hybridizes under stringent conditions to a polynucleotide specified in (a) encoding a polypeptide exhibiting the same biological function as specified for the respective sequence in Table 2 or 3;
 - (c) a polynucleotide the sequence of which deviates from the polynucleotide specified in (a) and (b) due to the generation of the genetic code encoding a polypeptide exhibiting the same biological function as specified for the respective sequence in Table 2 or 3;
 - 10 (d) a polynucleotide which represents a specific fragment, derivative or allelic variation of a polynucleotide sequence specified in (a) to (c);

in a biological sample comprising the following steps: hybridizing any polynucleotide or analogous oligomer specified in (a) to (d) to a polynucleotide material of a biological sample, thereby forming a hybridization complex; and detecting said hybridization complex.

15 In another embodiment the method for the prediction, diagnosis or prognosis of malignant neoplasia is done as just described but, wherein before hybridization, the polynucleotide material of the biological sample is amplified.

In another embodiment the method for the diagnosis or prognosis of malignant neoplasia and breast cancer in particular is done by the detection of:

- 20
- (a) a polynucleotide selected from the polynucleotides of the SEQ ID NO: 2 to 6, 8, 9, 11 to 16, 18, 19, 21 to 26 or 53 to 75;
 - (b) a polynucleotide which hybridizes under stringent conditions to a polynucleotide specified in (a) encoding a polypeptide exhibiting the same biological function as specified for the respective sequence in Table 2 or 3;
 - 25 (c) a polynucleotide the sequence of which deviates from the polynucleotide specified in (a) and (b) due to the generation of the genetic code encoding a polypeptide exhibiting the same biological function as specified for the respective sequence in Table 2 or 3;
 - (d) a polynucleotide which represents a specific fragment, derivative or allelic variation of a polynucleotide sequence specified in (a) to (c);
 - (e) a polypeptide encoded by a polynucleotide sequence specified in (a) to (d)

- (f) a polypeptide comprising any polypeptide of SEQ ID NO: 28 to 32, 34, 35, 37 to 42, 45, 47 to 52 or 76 to 98;

comprising the steps of contacting a biological sample with a reagent which specifically interacts with the polynucleotide specified in (a) to (d) or the polypeptide specified in (e).

5 DNA array technology

10 In one embodiment, the present Invention also provides a method wherein polynucleotide probes are immobilized on a DNA chip in an organized array. Oligonucleotides can be bound to a solid support by a variety of processes, including lithography. For example a chip can hold up to 4100,00 oligonucleotides (GeneChip, Affymetrix). The present invention provides significant advantages over the available tests for malignant neoplasia, such as breast cancer, because it increases the reliability of the test by providing an array of polynucleotide markers on a single chip.

15 The method includes obtaining a biopsy of an affected person, which is optionally fractionated by cryostat sectioning to enrich diseased cells to about 80% of the total cell population and the use of body fluids such as serum or urine, serum or cell containing liquids (e.g. derived from fine needle aspirates). The DNA or RNA is then extracted, amplified, and analyzed with a DNA chip to determine the presence or absence of the marker polynucleotide sequences. In one embodiment the polynucleotide probes are spotted onto a substrate in a two-dimensional matrix or array. Samples of polynucleotides can be labeled and then hybridized to the probes. Double-stranded polynucleotides, comprising the labeled sample polynucleotides bound to probe polynucleotides, can be detected once the unbound portion of the sample is washed away.

20 The probe polynucleotides can be spotted on substrates including glass, nitrocellulose, etc. The probes can be bound to the substrate by either covalent bonds or by non-specific interactions, such as hydrophobic interactions. The sample polynucleotides can be labeled using radioactive labels, fluorophores, chromophores, etc. Techniques for constructing arrays and methods of using these arrays are described in EP 0 799 897; WO 97/29212; WO 97/27317; EP 0 785 280; WO 97/02357; U.S. Pat. No. 5,593,839; U.S. Pat. No. 5,578,832; EP 0 728 520; U.S. Pat. No. 5,599,695; EP 0 721 016; U.S. Pat. No. 5,556,752; WO 95/22058; and U.S. Pat. No. 5,631,734. Further, arrays can be used to examine differential expression of genes and can be used to determine gene function. For example, arrays of the instant polynucleotide sequences can be used to determine if any of the polynucleotide sequences are differentially expressed between normal cells and diseased cells, for

example. High expression of a particular message in a diseased sample, which is not observed in a corresponding normal sample, can indicate a breast cancer specific protein.

Accordingly, in one aspect, the invention provides probes and primers that are specific to the unique polynucleotide markers disclosed herein.

5 In one embodiment, the method comprises using a polynucleotide probe to determine the presence of malignant or breast cancer cells in particular in a tissue from a patient. Specifically, the method comprises:

- 10 1) providing a polynucleotide probe comprising a nucleotide sequence at least 12 nucleotides in length, preferably at least 15 nucleotides, more preferably, 25 nucleotides, and most preferably at least 40 nucleotides, and up to all or nearly all of the coding sequence which is complementary to a portion of the coding sequence of a polynucleotide selected from the polynucleotides of SEQ ID NO: 1 to 26 and 53 to 75 or a sequence complementary thereto and is
- 2) differentially expressed in malignant neoplasia, such as breast cancer;
- 15 3) obtaining a tissue sample from a patient with malignant neoplasia;
- 4) providing a second tissue sample from a patient with no malignant neoplasia;
- 5) contacting the polynucleotide probe under stringent conditions with RNA of each of said first and second tissue samples (e.g., in a Northern blot or in situ hybridization assay); and
- 20 6) comparing (a) the amount of hybridization of the probe with RNA of the first tissue sample, with (b) the amount of hybridization of the probe with RNA of the second tissue sample;

wherein a statistically significant difference in the amount of hybridization with the RNA of the first tissue sample as compared to the amount of hybridization with the RNA of the second tissue sample is indicative of malignant neoplasia and breast cancer in particular in the first tissue sample.

25

Data analysis methods

Comparison of the expression levels of one or more "BREAST CANCER GENES" with reference expression levels, e.g., expression levels in diseased cells of breast cancer or in normal counterpart cells, is preferably conducted using computer systems. In one embodiment, expression levels are

obtained in two cells and these two sets of expression levels are introduced into a computer system for comparison. In a preferred embodiment, one set of expression levels is entered into a computer system for comparison with values that are already present in the computer system, or in a computer-readable form that is then entered into the computer system.

5 In one embodiment, the invention provides a computer readable form of the gene expression profile data of the invention, or of values corresponding to the level of expression of at least one "BREAST CANCER GENE" in a diseased cell. The values can be mRNA expression level obtained from experiments, e.g., microarray analysis. The values can also be mRNA level
10 normalised relative to a reference gene whose expression is constant in numerous cells under numerous conditions, e.g., GAPDH. In other embodiments, the values in the computer are ratios of, or differences between, normalized or non-normalized mRNA levels in different samples.

The gene expression profile data can be in the form of a table, such as an Excel table. The data can be alone, or it can be part of a larger database, e.g., comprising other expression profiles. For example, the expression profile data of the invention can be part of a public database. The
15 computer readable form can be in a computer. In another embodiment, the invention provides a computer displaying the gene expression profile data.

In one embodiment, the invention provides a method for determining the similarity between the level of expression of one or more "BREAST CANCER GENES" in a first cell, e.g., a cell of a subject, and that in a second cell, comprising obtaining the level of expression of one or more
20 "BREAST CANCER GENES" in a first cell and entering these values into a computer comprising a database including records comprising values corresponding to levels of expression of one or more "BREAST CANCER GENES" in a second cell, and processor instructions, e.g., a user interface, capable of receiving a selection of one or more values for comparison purposes with data that is stored in the computer. The computer may further comprise a means for converting the
25 comparison data into a diagram or chart or other type of output.

In another embodiment, values representing expression levels of "BREAST CANCER GENES" are entered into a computer system, comprising one or more databases with reference expression levels obtained from more than one cell. For example, the computer comprises expression data of diseased and normal cells. Instructions are provided to the computer, and the computer is capable
30 of comparing the data entered with the data in the computer to determine whether the data entered is more similar to that of a normal cell or of a diseased cell.

5 In another embodiment, the computer comprises values of expression levels in cells of subjects at different stages of breast cancer, and the computer is capable of comparing expression data entered into the computer with the data stored, and produce results indicating to which of the expression profiles in the computer, the one entered is most similar, such as to determine the stage of breast cancer in the subject.

10 In yet another embodiment, the reference expression profiles in the computer are expression profiles from cells of breast cancer of one or more subjects, which cells are treated *in vivo* or *in vitro* with a drug used for therapy of breast cancer. Upon entering of expression data of a cell of a subject treated *in vitro* or *in vivo* with the drug, the computer is instructed to compare the data entered to the data in the computer, and to provide results indicating whether the expression data input into the computer are more similar to those of a cell of a subject that is responsive to the drug or more similar to those of a cell of a subject that is not responsive to the drug. Thus, the results indicate whether the subject is likely to respond to the treatment with the drug or unlikely to respond to it.

15 In one embodiment, the invention provides a system that comprises a means for receiving gene expression data for one or a plurality of genes; a means for comparing the gene expression data from each of said one or plurality of genes to a common reference frame; and a means for presenting the results of the comparison. This system may further comprise a means for clustering the data.

20 In another embodiment, the invention provides a computer program for analyzing gene expression data comprising (i) a computer code that receives as input gene expression data for a plurality of genes and (ii) a computer code that compares said gene expression data from each of said plurality of genes to a common reference frame.

25 The invention also provides a machine-readable or computer-readable medium including program instructions for performing the following steps: (i) comparing a plurality of values corresponding to expression levels of one or more genes characteristic of breast cancer in a query cell with a database including records comprising reference expression or expression profile data of one or more reference cells and an annotation of the type of cell; and (ii) indicating to which cell the query cell is most similar based on similarities of expression profiles. The reference cells can be 30 cells from subjects at different stages of breast cancer. The reference cells can also be cells from subjects responding or not responding to a particular drug treatment and optionally incubated *in vitro* or *in vivo* with the drug.

The reference cells may also be cells from subjects responding or not responding to several different treatments, and the computer system indicates a preferred treatment for the subject. Accordingly, the invention provides a method for selecting a therapy for a patient having breast cancer, the method comprising: (i) providing the level of expression of one or more genes characteristic of breast cancer in a diseased cell of the patient; (ii) providing a plurality of reference profiles, each associated with a therapy, wherein the subject expression profile and each reference profile has a plurality of values, each value representing the level of expression of a gene characteristic of breast cancer; and (iii) selecting the reference profile most similar to the subject expression profile, to thereby select a therapy for said patient. In a preferred embodiment step (ii) is performed by a computer. The most similar reference profile may be selected by weighing the comparison value of the plurality using a weight value associated with the corresponding expression data.

The relative abundance of an mRNA in two biological samples can be scored as a perturbation and its magnitude determined (i.e., the abundance is different in the two sources of mRNA tested), or as not perturbed (i.e., the relative abundance is the same). In various embodiments, a difference between the two sources of RNA of at least a factor of about 25% (RNA from one source is 25% more abundant in one source than the other source), more usually about 50%, even more often by a factor of about 2 (twice as abundant), 3 (three times as abundant) or 5 (five times as abundant) is scored as a perturbation. Perturbations can be used by a computer for calculating and expression comparisons.

Preferably, in addition to identifying a perturbation as positive or negative, it is advantageous to determine the magnitude of the perturbation. This can be carried out, as noted above, by calculating the ratio of the emission of the two fluorophores used for differential labeling, or by analogous methods that will be readily apparent to those of skill in the art.

The computer readable medium may further comprise a pointer to a descriptor of a stage of breast cancer or to a treatment for breast cancer.

In operation, the means for receiving gene expression data, the means for comparing the gene expression data, the means for presenting, the means for normalizing, and the means for clustering within the context of the systems of the present invention can involve a programmed computer with the respective functionalities described herein, implemented in hardware or hardware and software; a logic circuit or other component of a programmed computer that performs the operations specifically identified herein, dictated by a computer program; or a computer memory

encoded with executable instructions representing a computer program that can cause a computer to function in the particular fashion described herein.

Those skilled in the art will understand that the systems and methods of the present invention may be applied to a variety of systems, including IBM-compatible personal computers running MS-DOS or Microsoft Windows.

The computer may have internal components linked to external components. The internal components may include a processor element interconnected with a main memory. The computer system can be an Intel Pentium®-based processor of 200 MHz or greater clock rate and with 32 MB or more of main memory. The external component may comprise a mass storage, which can be one or more hard disks (which are typically packaged together with the processor and memory). Such hard disks are typically of 1 GB or greater storage capacity. Other external components include a user interface device, which can be a monitor, together with an inputting device, which can be a "mouse", or other graphic input devices, and/or a keyboard. A printing device can also be attached to the computer.

Typically, the computer system is also linked to a network link, which can be part of an Ethernet link to other local computer systems, remote computer systems, or wide area communication networks, such as the Internet. This network link allows the computer system to share data and processing tasks with other computer systems.

Loaded into memory during operation of this system are several software components, which are both standard in the art and special to the instant invention. These software components collectively cause the computer system to function according to the methods of this invention. These software components are typically stored on a mass storage. A software component represents the operating system, which is responsible for managing the computer system and its network interconnections. This operating system can be, for example, of the Microsoft Windows' family, such as Windows 95, Windows 98, or Windows NT. A software component represents common languages and functions conveniently present on this system to assist programs implementing the methods specific to this invention. Many high or low level computer languages can be used to program the analytic methods of this invention. Instructions can be interpreted during run-time or compiled. Preferred languages include C/C++, and JAVA®. Most preferably, the methods of this invention are programmed in mathematical software packages which allow symbolic entry of equations and high-level specification of processing, including algorithms to be used, thereby freeing a user of the need to procedurally program individual equations or algorithms. Such packages include Matlab from Mathworks (Natick, Mass.), Mathematica from

Wolfram Research (Champaign, Ill.), or S-Plus from Math Soft (Cambridge, Mass.). Accordingly, a software component represents the analytic methods of this invention as programmed in procedural language or symbolic package. In a preferred embodiment, the computer system also contains a database comprising values representing levels of expression of one or more gene characteristic of breast cancer. The database may contain one or more expression profiles of gene characteristic of breast cancer in different cells.

In an exemplary implementation, to practice the methods of the present invention, a user first loads expression profile data into the computer system. These data can be directly entered by the user from a monitor and keyboard, or from other computer systems linked by a network connection, or on removable storage media such as a CD-ROM or floppy disk or through the network. Next the user causes execution of expression profile analysis software which performs the steps of comparing and, e.g., clustering co-varying genes into groups of genes.

In another exemplary implementation, expression profiles are compared using a method described in U.S. Patent No. 6,203,987. A user first loads expression profile data into the computer system. Geneset profile definitions are loaded into the memory from the storage media or from a remote computer, preferably from a dynamic geneset database system, through the network. Next the user causes execution of projection software which performs the steps of converting expression profile to projected expression profiles. The projected expression profiles are then displayed.

In yet another exemplary implementation, a user first loads a projected profile into the memory. The user then causes the loading of a reference profile into the memory. Next, the user causes the execution of comparison software which performs the steps of objectively comparing the profiles.

Detection of variant polynucleotide sequence

In yet another embodiment, the invention provides methods for determining whether a subject is at risk for developing a disease, such as a predisposition to develop malignant neoplasia, for example breast cancer, associated with an aberrant activity of any one of the polypeptides encoded by any of the polynucleotides of the SEQ ID NO: 1 to 26 or 53 to 75, wherein the aberrant activity of the polypeptide is characterized by detecting the presence or absence of a genetic lesion characterized by at least one of these:

- (i) an alteration affecting the integrity of a gene encoding a marker polypeptides, or
- (ii) the misexpression of the encoding polynucleotide.

To illustrate, such genetic lesions can be detected by ascertaining the existence of at least one of these:

- I. a deletion of one or more nucleotides from the polynucleotide sequence
- II. an addition of one or more nucleotides to the polynucleotide sequence
- 5 III. a substitution of one or more nucleotides of the polynucleotide sequence
- IV. a gross chromosomal rearrangement of the polynucleotide sequence
- V. a gross alteration in the level of a messenger RNA transcript of the polynucleotide sequence
- VI. aberrant modification of the polynucleotide sequence, such as of the methylation pattern
10 of the genomic DNA
- VII. the presence of a non-wild type splicing pattern of a messenger RNA transcript of the gene
- VIII. a non-wild type level of the marker polypeptide
- IX. allelic loss of the gene
- X. allelic gain of the gene
- 15 XI. inappropriate post-translational modification of the marker polypeptide

The present Invention provides assay techniques for detecting mutations in the encoding polynucleotide sequence. These methods include, but are not limited to, methods involving sequence analysis, Southern blot hybridization, restriction enzyme site mapping, and methods involving detection of absence of nucleotide pairing between the polynucleotide to be analyzed
20 and a probe.

Specific diseases or disorders, e.g., genetic diseases or disorders, are associated with specific allelic variants of polymorphic regions of certain genes, which do not necessarily encode a mutated protein. Thus, the presence of a specific allelic variant of a polymorphic region of a gene in a subject can render the subject susceptible to developing a specific disease or disorder.
25 Polymorphic regions in genes, can be identified, by determining the nucleotide sequence of genes in populations of individuals. If a polymorphic region is identified, then the link with a specific disease can be determined by studying specific populations of individuals, e.g. individuals which developed a specific disease, such as breast cancer. A polymorphic region can be located in any

region of a gene, e.g., exons, in coding or non coding regions of exons, introns, and promote region.

5 In an exemplary embodiment, there is provided a polynucleotide composition comprising a polynucleotide probe including a region of nucleotide sequence which is capable of hybridising to a sense or antisense sequence of a gene or naturally occurring mutants thereof, or 5' or 3' flanking sequences or intronic sequences naturally associated with the subject genes or naturally occurring mutants thereof. The polynucleotide of a cell is rendered accessible for hybridization, the probe is contacted with the polynucleotide of the sample, and the hybridization of the probe to the sample polynucleotide is detected. Such techniques can be used to detect lesions or allelic variants at
10 either the genomic or mRNA level, including deletions, substitutions, etc., as well as to determine mRNA transcript levels.

A preferred detection method is allele specific hybridization using probes overlapping the mutation or polymorphic site and having about 5, 10, 20, 25, or 30 nucleotides around the mutation or polymorphic region. In a preferred embodiment of the invention, several probes
15 capable of hybridising specifically to allelic variants are attached to a solid phase support, e.g., a "chip". Mutation detection analysis using these chips comprising oligonucleotides, also termed "DNA probe arrays" is described e.g., in Cronin et al. (119). In one embodiment, a chip comprises all the allelic variants of at least one polymorphic region of a gene. The solid phase support is then contacted with a test polynucleotide and hybridization to the specific probes is detected.
20 Accordingly, the identity of numerous allelic variants of one or more genes can be identified in a simple hybridization experiment.

In certain embodiments, detection of the lesion comprises utilizing the probe/primer in a polymerase chain reaction (PCR) (see, e.g. U.S. Patent Nos. 4,683,195 and 4,683,202), such as anchor PCR or RACE PCR, or, alternatively, in a ligase chain reaction (LCR) [Landegran et al.,
25 1988, (120) and Nakazawa et al., 1994 (121)], the latter of which can be particularly useful for detecting point mutations in the gene; Abravaya et al., 1995 ,(122)]. In a merely illustrative embodiment, the method includes the steps of (i) collecting a sample of cells from a patient, (ii) isolating polynucleotide (e.g., genomic, mRNA or both) from the cells of the sample, (iii) contacting the polynucleotide sample with one or more primers which specifically hybridize to a
30 polynucleotide sequence under conditions such that hybridization and amplification of the polynucleotide (if present) occurs, and (iv) detecting the presence or absence of an amplification product, or detecting the size of the amplification product and comparing the length to a control sample. It is anticipated that PCR and/or LCR may be desirable to use as a preliminary

amplification step in conjunction with any of the techniques used for detecting mutations described herein.

Alternative amplification methods include: self sustained sequence replication [Guatelli, J.C. et al., 1990, (123)], transcriptional amplification system [Kwoh, D.Y. et al., 1989, (124)], Q-Beta replicase [Lizardi, P.M. et al., 1988, (125)], or any other polynucleotide amplification method, followed by the detection of the amplified molecules using techniques well known to those of skill in the art. These detection schemes are especially useful for the detection of polynucleotide molecules if such molecules are present in very low numbers.

In a preferred embodiment of the subject assay, mutations in, or allelic variants, of a gene from a sample cell are identified by alterations in restriction enzyme cleavage patterns. For example, sample and control DNA is isolated, amplified (optionally), digested with one or more restriction endonucleases, and fragment length sizes are determined by gel electrophoresis. Moreover, the use of sequence specific ribozymes (see, for example, U.S. Patent No. 5,498,531) can be used to score for the presence of specific mutations by development or loss of a ribozyme cleavage site.

In situ hybridization

In one aspect, the method comprises *in situ* hybridization with a probe derived from a given marker polynucleotide, which sequence is selected from any of the polynucleotide sequences of the SEQ ID NO: 1 to 9, or 11 to 19 or 21 to 26 and 53 to 75 or a sequence complementary thereto. The method comprises contacting the labeled hybridization probe with a sample of a given type of tissue from a patient potentially having malignant neoplasia and breast cancer in particular as well as normal tissue from a person with no malignant neoplasia, and determining whether the probe labels tissue of the patient to a degree significantly different (e.g., by at least a factor of two, or at least a factor of five, or at least a factor of twenty, or at least a factor of fifty) than the degree to which normal tissue is labelled.

Polypeptide detection

The subject invention further provides a method of determining whether a cell sample obtained from a subject possesses an abnormal amount of marker polypeptide which comprises (a) obtaining a cell sample from the subject, (b) quantitatively determining the amount of the marker polypeptide in the sample so obtained, and (c) comparing the amount of the marker polypeptide so determined with a known standard, so as to thereby determine whether the cell sample obtained from the subject possesses an abnormal amount of the marker polypeptide. Such marker

polypeptides may be detected by immunohistochemical assays, dot-blot assays, ELISA and the like.

Antibodies

5 Any type of antibody known in the art can be generated to bind specifically to an epitope of a „BREAST CANCER GENE“ polypeptide. An antibody as used herein includes intact immunoglobulin molecules, as well as fragments thereof, such as Fab, F(ab)₂, and Fv, which are capable of binding an epitope of a „BREAST CANCER GENE“ polypeptide. Typically, at least 6, 8, 10, or 12 contiguous amino acids are required to form an epitope. However, epitopes which involve non-contiguous amino acids may require more, e.g., at least 15, 25, or 50 amino acids.

10 An antibody which specifically binds to an epitope of a „BREAST CANCER GENE“ polypeptide can be used therapeutically, as well as in immunochemical assays, such as Western blots, ELISAs, radioimmunoassays, immunohistochemical assays, immunoprecipitations, or other immunochemical assays known in the art. Various immunoassays can be used to identify antibodies having the desired specificity. Numerous protocols for competitive binding or
15 immunoradiometric assays are well known in the art. Such immunoassays typically involve the measurement of complex formation between an immunogen and an antibody which specifically binds to the immunogen.

Typically, an antibody which specifically binds to a „BREAST CANCER GENE“ polypeptide provides a detection signal at least 5-, 10-, or 20-fold higher than a detection signal provided with
20 other proteins when used in an immunochemical assay. Preferably, antibodies which specifically bind to „BREAST CANCER GENE“ polypeptides do not detect other proteins in immunochemical assays and can immunoprecipitate a „BREAST CANCER GENE“ polypeptide from solution.

„BREAST CANCER GENE“ polypeptides can be used to immunize a mammal, such as a mouse,
25 rat, rabbit, guinea pig, monkey, or human, to produce polyclonal antibodies. If desired, a „BREAST CANCER GENE“ polypeptide can be conjugated to a carrier protein, such as bovine serum albumin, thyroglobulin, and keyhole limpet hemocyanin. Depending on the host species, various adjuvants can be used to increase the immunological response. Such adjuvants include, but are not limited to, Freund's adjuvant, mineral gels (e.g., aluminum hydroxide), and surface
30 active substances (e.g. lysolecithin, pluronic polyols, polyanions, peptides, oil emulsions, keyhole limpet hemocyanin, and dinitrophenol). Among adjuvants used in humans, BCG (bacilli Calmette-Guerin) and Corynebacterium parvum are especially useful.

Monoclonal antibodies which specifically bind to a „BREAST CANCER GENE“ polypeptide can be prepared using any technique which provides for the production of antibody molecules by continuous cell lines in culture. These techniques include, but are not limited to, the hybridoma technique, the human B cell hybridoma technique, and the EBV hybridoma technique [Kohler et al., 1985, (136); Kozbor et al., 1985, (137); Cote et al., 1983, (138) and Cole et al., 1984, (139)].

In addition, techniques developed for the production of chimeric antibodies, the splicing of mouse antibody genes to human antibody genes to obtain a molecule with appropriate antigen specificity and biological activity, can be used [Morrison et al., 1984, (140); Neuberger et al., 1984, (141); Takeda et al., 1985, (142)]. Monoclonal and other antibodies also can be humanized to prevent a patient from mounting an immune response against the antibody when it is used therapeutically. Such antibodies may be sufficiently similar in sequence to human antibodies to be used directly in therapy or may require alteration of a few key residues. Sequence differences between rodent antibodies and human sequences can be minimized by replacing residues which differ from those in the human sequences by site directed mutagenesis of individual residues or by grafting of entire complementarity determining regions. Alternatively, humanized antibodies can be produced using recombinant methods, as described in GB2188638B. Antibodies which specifically bind to a „BREAST CANCER GENE“ polypeptide can contain antigen binding sites which are either partially or fully humanized, as disclosed in U.S. Patent 5,565,332.

Alternatively, techniques described for the production of single chain antibodies can be adapted using methods known in the art to produce single chain antibodies which specifically bind to „BREAST CANCER GENE“ polypeptides. Antibodies with related specificity, but of distinct idiotypic composition, can be generated by chain shuffling from random combinatorial immunoglobulin libraries [Burton, 1991, (143)].

Single-chain antibodies also can be constructed using a DNA amplification method, such as PCR, using hybridoma cDNA as a template [Thirion et al., 1996, (144)]. Single-chain antibodies can be mono- or bispecific, and can be bivalent or tetravalent. Construction of tetravalent, bispecific single-chain antibodies is taught, for example, in Coloma & Morrison, (145). Construction of bivalent, bispecific single-chain antibodies is taught in Mallender & Voss, (146).

A nucleotide sequence encoding a single-chain antibody can be constructed using manual or automated nucleotide synthesis, cloned into an expression construct using standard recombinant DNA methods, and introduced into a cell to express the coding sequence, as described below. Alternatively, single-chain antibodies can be produced directly using, for example, filamentous phage technology [Verhaar et al., 1995, (147); Nicholls et al., 1993, (148)].

Antibodies which specifically bind to „BREAST CANCER GENE“ polypeptides also can be produced by inducing in vivo production in the lymphocyte population or by screening immunoglobulin libraries or panels of highly specific binding reagents as disclosed in the literature [Orlandi et al., 1989, (149) and Winter et al., 1991, (150)].

5 Other types of antibodies can be constructed and used therapeutically in methods of the invention. For example, chimeric antibodies can be constructed as disclosed in WO 93/03151. Binding proteins which are derived from immunoglobulins and which are multivalent and multispecific such as the antibodies described in WO 94/13804, also can be prepared.

10 Antibodies according to the invention can be purified by methods well known in the art. For example, antibodies can be affinity purified by passage over a column to which a „BREAST CANCER GENE“ polypeptide is bound. The bound antibodies can then be eluted from the column using a buffer with a high salt concentration.

15 Immunoassays are commonly used to quantify the levels of proteins in cell samples, and many other immunoassay techniques are known in the art. The invention is not limited to a particular assay procedure, and therefore is intended to include both homogeneous and heterogeneous procedures. Exemplary immunoassays which can be conducted according to the invention include fluorescence polarisation immunoassay (FPIA), fluorescence immunoassay (FIA), enzyme immunoassay (EIA), nephelometric inhibition immunoassay (NIA), enzyme linked immunosorbent assay (ELISA), and radioimmunoassay (RIA). An indicator moiety, or label group, can be attached to the subject antibodies and is selected so as to meet the needs of various uses of the method which are often dictated by the availability of assay equipment and compatible immunoassay procedures. General techniques to be used in performing the various immunoassays noted above are known to those of ordinary skill in the art.

25 In another embodiment, the level of at least one product encoded by any of the polynucleotide sequences of the SEQ ID NO: 2 to 6, 8, 9, 11 to 16, 18, 19 or 21 to 26 or 53 to 75 or of at least 2 products encoded by a polynucleotide selected from SEQ ID NO: 1 to 26 and 53 to 75 or a sequence complementary thereto, in a biological fluid (e.g., blood or urine) of a patient may be determined as a way of monitoring the level of expression of the marker polynucleotide sequence in cells of that patient. Such a method would include the steps of obtaining a sample of a biological fluid from the patient, contacting the sample (or proteins from the sample) with an antibody specific for a encoded marker polypeptide, and determining the amount of immune complex formation by the antibody, with the amount of immune complex formation being indicative of the level of the marker encoded product in the sample. This determination is

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particularly instructive when compared to the amount of immune complex formation by the same antibody in a control sample taken from a normal individual or in one or more samples previously or subsequently obtained from the same person.

5 In another embodiment, the method can be used to determine the amount of marker polypeptide present in a cell, which in turn can be correlated with progression of the disorder, e.g., plaque formation. The level of the marker polypeptide can be used predictively to evaluate whether a sample of cells contains cells which are, or are predisposed towards becoming, plaque associated cells. The observation of marker polypeptide level can be utilized in decisions regarding, e.g., the use of more stringent therapies.

10 As set out above, one aspect of the present invention relates to diagnostic assays for determining, in the context of cells isolated from a patient, if the level of a marker polypeptide is significantly reduced in the sample cells. The term "significantly reduced" refers to a cell phenotype wherein the cell possesses a reduced cellular amount of the marker polypeptide relative to a normal cell of similar tissue origin. For example, a cell may have less than about 50%, 25%, 10%, or 5% of the
15 marker polypeptide that a normal control cell. In particular, the assay evaluates the level of marker polypeptide in the test cells, and, preferably, compares the measured level with marker polypeptide detected in at least one control cell, e.g., a normal cell and/or a transformed cell of known phenotype.

Of particular importance to the subject invention is the ability to quantify the level of marker
20 polypeptide as determined by the number of cells associated with a normal or abnormal marker polypeptide level. The number of cells with a particular marker polypeptide phenotype may then be correlated with patient prognosis. In one embodiment of the invention, the marker polypeptide phenotype of the lesion is determined as a percentage of cells in a biopsy which are found to have abnormally high/low levels of the marker polypeptide. Such expression may be detected by
25 immunohistochemical assays, dot-blot assays, ELISA and the like.

Immunohistochemistry

Where tissue samples are employed, immunohistochemical staining may be used to determine the number of cells having the marker polypeptide phenotype. For such staining, a multiblock of tissue is taken from the biopsy or other tissue sample and subjected to proteolytic hydrolysis,
30 employing such agents as protease K or pepsin. In certain embodiments, it may be desirable to isolate a nuclear fraction from the sample cells and detect the level of the marker polypeptide in the nuclear fraction.

5 The tissues samples are fixed by treatment with a reagent such as formalin, glutaraldehyde, methanol, or the like. The samples are then incubated with an antibody, preferably a monoclonal antibody, with binding specificity for the marker polypeptides. This antibody may be conjugated to a Label for subsequent detection of binding. samples are incubated for a time Sufficient for formation of the immunocomplexes. Binding of the antibody is then detected by virtue of a Label conjugated to this antibody. Where the antibody is unlabelled, a second labeled antibody may be employed, e.g., which is specific for the isotype of the anti-marker polypeptide antibody. Examples of labels which may be employed include radionuclides, fluorescence, chemiluminescence, and enzymes.

10 Where enzymes are employed, the Substrate for the enzyme may be added to the samples to provide a colored or fluorescent product. Examples of suitable enzymes for use in conjugates include horseradish peroxidase, alkaline phosphatase, malate dehydrogenase and the like. Where not commercially available, such antibody-enzyme conjugates are readily produced by techniques known to those skilled in the art.

15 In one embodiment, the assay is performed as a dot blot assay. The dot blot assay finds particular application where tissue samples are employed as it allows determination of the average amount of the marker polypeptide associated with a Single cell by correlating the amount of marker polypeptide in a cell-free extract produced from a predetermined number of cells.

20 In yet another embodiment, the invention contemplates using one or more antibodies which are generated against one or more of the marker polypeptides of this invention, which polypeptides are encoded by any of the polynucleotide sequences of the SEQ ID NO: 1 to 26 or 53 to 75. Such a panel of antibodies may be used as a reliable diagnostic probe for breast cancer. The assay of the present invention comprises contacting a biopsy sample containing cells, e.g., macrophages, with a panel of antibodies to one or more of the encoded products to determine the presence or absence of the marker polypeptides.

25 The diagnostic methods of the subject invention may also be employed as follow-up to treatment, e.g., quantification of the level of marker polypeptides may be indicative of the effectiveness of current or previously employed therapies for malignant neoplasia and breast cancer in particular as well as the effect of these therapies upon patient prognosis.

30 The diagnostic assays described above can be adapted to be used as prognostic assays, as well. Such an application takes advantage of the sensitivity of the assays of the Invention to events which take place at characteristic stages in the progression of plaque generation in case of

malignant neoplasia. For example, a given marker gene may be up- or down-regulated at a very early stage, perhaps before the cell is developing into a foam cell, while another marker gene may be characteristically up or down regulated only at a much later stage. Such a method could involve the steps of contacting the mRNA of a test cell with a polynucleotide probe derived from a given marker polynucleotide which is expressed at different characteristic levels in breast cancer tissue cells at different stages of malignant neoplasia progression, and determining the approximate amount of hybridization of the probe to the mRNA of the cell, such amount being an indication of the level of expression of the gene in the cell, and thus an indication of the stage of disease progression of the cell; alternatively, the assay can be carried out with an antibody specific for the gene product of the given marker polynucleotide, contacted with the proteins of the test cell. A battery of such tests will disclose not only the existence of a certain arteriosclerotic plaque, but also will allow the clinician to select the mode of treatment most appropriate for the disease, and to predict the likelihood of success of that treatment.

The methods of the invention can also be used to follow the clinical course of a given breast cancer predisposition. For example, the assay of the Invention can be applied to a blood sample from a patient; following treatment of the patient for BREAST CANCER, another blood sample is taken and the test repeated. Successful treatment will result in removal of demonstrate differential expression, characteristic of the breast cancer tissue cells, perhaps approaching or even surpassing normal levels.

Polypeptide activity

In one embodiment the present invention provides a method for screening potentially therapeutic agents which modulate the activity of one or more "BREAST CANCER GENE" polypeptides, such that if the activity of the polypeptide is increased as a result of the upregulation of the "BREAST CANCER GENE" in a subject having or at risk for malignant neoplasia and breast cancer in particular, the therapeutic substance will decrease the activity of the polypeptide relative to the activity of the some polypeptide in a subject not having or not at risk for malignant neoplasia or breast cancer in particular but not treated with the therapeutic agent. Likewise, if the activity of the polypeptide as a result of the downregulation of the "BREAST CANCER GENE" is decreased in a subject having or at risk for malignant neoplasia or breast cancer in particular, the therapeutic agent will increase the activity of the polypeptide relative to the activity of the same polypeptide in a subject not having or not at risk for malignant neoplasia or breast cancer in particular, but not treated with the therapeutic agent.

The activity of the "BREAST CANCER GENE" polypeptides indicated in Table 2 or 3 may be measured by any means known to those of skill in the art, and which are particular for the type of activity performed by the particular polypeptide. Examples of specific assays which may be used to measure the activity of particular polynucleotides are shown below.

5 a) G protein coupled receptors

In one embodiment, the "BREAST CANCER GENE" polynucleotide may encode a G protein coupled receptor. In one embodiment, the present invention provides a method of screening potential modulators (inhibitors or activators) of the G protein coupled receptor by measuring changes in the activity of the receptor in the presence of a candidate modulator.

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1. G_i -coupled receptors

Cells (such as CHO cells or primary cells) are stably transfected with the relevant receptor and with an inducible CRE-luciferase construct. Cells are grown in 50% Dulbecco's modified Eagle medium / 50% F12 (DMEM/F12) supplemented with 10% FBS, at 37°C in a humidified atmosphere with 10% CO₂ and are routinely split at a ratio of 1:10 every 2 or 3 days. Test cultures are seeded into 384 - well plates at an appropriate density (e.g. 2000 cells / well in 35 µl cell culture medium) in DMEM/F12 with FBS, and are grown for 48 hours (range: ~ 24 - 60 hours, depending on cell line). Growth medium is then exchanged against serum free medium (SFM; e.g. Ultra-CHO), containing 0,1% BSA. Test compounds dissolved in DMSO are diluted in SFM and transferred to the test cultures (maximal final concentration 10 µmolar), followed by addition of forskolin (~ 1 µmolar, final conc.) in SFM + 0,1% BSA 10 minutes later. In case of antagonist screening both, an appropriate concentration of agonist, and forskolin are added. The plates are incubated at 37°C in 10% CO₂ for 3 hours. Then the supernatant is removed, cells are lysed with lysis reagent (25 mmolar phosphate-buffer, pH 7,8, containing 2 mmolar DDT, 10% glycerol and 3% Triton X100). The luciferase reaction is started by addition of substrate-buffer (e.g. luciferase assay reagent, Promega) and luminescence is immediately determined (e.g. Berthold luminometer or Hamamatzu camera system).

2. G_s -coupled receptors

Cells (such as CHO cells or primary cells) are stably transfected with the relevant receptor and with an inducible CRE-luciferase construct. Cells are grown in 50% Dulbecco's modified Eagle medium / 50% F12 (DMEM/F12) supplemented with 10% FBS, at 37°C in a humidified

atmosphere with 10% CO₂ and are routinely split at a ratio of 1:10 every 2 or 3 days. Test cultures are seeded into 384 – well plates at an appropriate density (e.g. 1000 or 2000 cells / well in 35 µl cell culture medium) in DMEM/F12 with FBS, and are grown for 48 hours (range: ~ 24 - 60 hours, depending on cell line). The assay is started by addition of test-compounds in serum free medium (SFM; e.g. Ultra-CHO) containing 0,1% BSA: Test compounds are dissolved in DMSO, diluted in SFM and transferred to the test cultures (maximal final concentration 10 µmolar, DMSO conc. < 0,6 %). In case of antagonist screening an appropriate concentration of agonist is added 5 – 10 minutes later. The plates are incubated at 37°C in 10% CO₂ for 3 hours. Then the cells are lysed with 10 µl lysis reagent per well (25 mmolar phosphate-buffer, pH 7,8 , containing 2 mmolar DDT, 10% glycerol and 3% Triton X100) and the luciferase reaction is started by addition of 20 µl substrate-buffer per well (e.g. luciferase assay reagent, Promega). Measurement of luminescence is started immediately (e.g. Berthold luminometer or Hamamatsu camera system).

3. G_q-coupled receptors

Cells (such as CHO cells or primary cells) are stably transfected with the relevant receptor. Cells expressing functional receptor protein are grown in 50% Dulbecco's modified Eagle medium / 50% F12 (DMEM/F12) supplemented with 10% FBS, at 37°C in a humidified atmosphere with 5% CO₂ and are routinely split at a cell line dependent ratio every 3 or 4 days. Test cultures are seeded into 384 – well plates at an appropriate density (e.g. 2000 cells / well in 35 µl cell culture medium) in DMEM/F12 with FBS, and are grown for 48 hours (range: ~ 24 - 60 hours, depending on cell line). Growth medium is then exchanged against physiological salt solution (e.g. Tyrode solution). Test compounds dissolved in DMSO are diluted in Tyrode solution containing 0.1% BSA and transferred to the test cultures (maximal final concentration 10 µmolar). After addition of the receptor specific agonist the resulting G_q-mediated intracellular calcium increase is measured using appropriate read-out systems (e.g. calcium-sensitive dyes).

b) Ion channels

Ion channels are integral membrane proteins involved in electrical signaling, transmembrane signal transduction, and electrolyte and solute transport. By forming macromolecular pores through the membrane lipid bilayer, ion channels account for the flow of specific ion species driven by the electrochemical potential gradient for the permeating ion. At the single molecule level, individual channels undergo conformational transitions ("gating") between the 'open' (ion conducting) and 'closed' (non conducting) state. Typical single channel openings last for a few milliseconds and result in elementary transmembrane currents in the range of 10⁻⁹ - 10⁻¹² Ampere. Channel gating is controlled by various chemical and/or biophysical parameters, such as

neurotransmitters and intracellular second messengers ('ligand-gated' channels) or membrane potential ('voltage-gated' channels). Ion channels are functionally characterized by their ion selectivity, gating properties, and regulation by hormones and pharmacological agents. Because of their central role in signaling and transport processes, ion channels present ideal targets for pharmacological therapeutics in various pathophysiological settings.

In one embodiment, the "BREAST CANCER GENE" may encode an ion channel. In one embodiment, the present invention provides a method of screening potential activators or inhibitors of channels activity of the "BREAST CANCER GENE" polypeptide. Screening for compounds interaction with ion channels to either inhibit or promote their activity can be based on (1.) binding and (2.) functional assays in living cells[Hille (183)].

1. For ligand-gated channels, e.g. ionotropic neurotransmitter/hormone receptors, assays can be designed detecting binding to the target by competition between the compound and a labeled ligand.
2. Ion channel function can be tested functionally in living cells. Target proteins are either expressed endogenously in appropriate reporter cells or are introduced recombinantly. Channel activity can be monitored by (2.1) concentration changes of the permeating ion (most prominently Ca^{2+} ions), (2.2) by changes in the transmembrane electrical potential gradient, and (2.3) by measuring a cellular response (e.g. expression of a reporter gene, secretion of a neurotransmitter) triggered or modulated by the target activity.
 - 2.1 Channel activity results in transmembrane ion fluxes. Thus activation of ionic channels can be monitored by the resulting changes in intracellular ion concentrations using luminescent or fluorescent indicators. Because of its wide dynamic range and availability of suitable indicators this applies particularly to changes in intracellular Ca^{2+} ion concentration ($[\text{Ca}^{2+}]_i$). $[\text{Ca}^{2+}]_i$ can be measured, for example, by aequorin luminescence or fluorescence dye technology (e.g. using Fluo-3, Indo-1, Fura-2). Cellular assays can be designed where either the Ca^{2+} flux through the target channel itself is measured directly or where modulation of the target channel affects membrane potential and thereby the activity of co-expressed voltage-gated Ca^{2+} channels.
 - 2.2 Ion channel currents result in changes of electrical membrane potential (V_m) which can be monitored directly using potentiometric fluorescent probes. These electrically charged indicators (e.g. the anionic oxonol dye DiBAC₄(3)) redistribute between extra- and

intracellular compartment in response to voltage changes. The equilibrium distribution is governed by the Nernst-equation. Thus changes in membrane potential results in concomitant changes in cellular fluorescence. Again, changes in V_m might be caused directly by the activity of the target ion channel or through amplification and/or prolongation of the signal by channels co-expressed in the same cell.

2.3 Target channel activity can cause cellular Ca^{2+} entry either directly or through activation of additional Ca^{2+} channel (see 2.1). The resulting intracellular Ca^{2+} signals regulate a variety of cellular responses, e.g. secretion or gene transcription. Therefore modulation of the target channel can be detected by monitoring secretion of a known hormone/transmitter from the target-expressing cell or through expression of a reporter gene (e.g. luciferase) controlled by an Ca^{2+} -responsive promoter element (e.g. cyclic AMP/ Ca^{2+} -responsive elements; CRE).

c) DNA-binding proteins and transcription factors

In one embodiment, the "BREAST CANCER GENE" may encode a DNA-binding protein or a transcription factor. The activity of such a DNA-binding protein or a transcription factor may be measured, for example, by a promoter assay which measures the ability of the DNA-binding protein or the transcription factor to initiate transcription of a test sequence linked to a particular promoter. In one embodiment, the present invention provides a method of screening test compounds for its ability to modulate the activity of such a DNA-binding protein or a transcription factor by measuring the changes in the expression of a test gene which is regulated by a promoter which is responsive to the transcription factor.

d) Promotor assays

A promoter assay was set up with a human hepatocellular carcinoma cell HepG2 that was stably transfected with a luciferase gene under the control of a gene of interest (e.g. thyroid hormone) regulated promoter. The vector 2xIROluc, which was used for transfection, carries a thyroid hormone responsive element (TRE) of two 12 bp inverted palindromes separated by an 8 bp spacer in front of a tk minimal promoter and the luciferase gene. Test cultures were seeded in 96 well plates in serum - free Eagle's Minimal Essential Medium supplemented with glutamine, tricine, sodium pyruvate, non - essential amino acids, insulin, selen, transferrin, and were cultivated in a humidified atmosphere at 10 % CO_2 at 37°C. After 48 hours of incubation serial dilutions of test compounds or reference compounds (L-T3, L-T4 e.g.) and co-stimulator if appropriate (final concentration 1 nM) were added to the cell cultures and incubation was continued for the optimal

time (e.g. another 4-72 hours). The cells were then lysed by addition of buffer containing Triton X100 and luciferin and the luminescence of luciferase induced by T3 or other compounds was measured in a luminometer. For each concentration of a test compound replicates of 4 were tested. EC₅₀ – values for each test compound were calculated by use of the Graph Pad Prism Scientific software.

Screening Methods

The invention provides assays for screening test compounds which bind to or modulate the activity of a „BREAST CANCER GENE“ polypeptide or a „BREAST CANCER GENE“ polynucleotide. A test compound preferably binds to a „BREAST CANCER GENE“ polypeptide or polynucleotide. More preferably, a test compound decreases or increases „BREAST CANCER GENE“ activity by at least about 10, preferably about 50, more preferably about 75, 90, or 100% relative to the absence of the test compound.

Test Compounds

Test compounds can be pharmacological agents already known in the art or can be compounds previously unknown to have any pharmacological activity. The compounds can be naturally occurring or designed in the laboratory. They can be isolated from microorganisms, animals, or plants, and can be produced recombinant, or synthesised by chemical methods known in the art. If desired, test compounds can be obtained using any of the numerous combinatorial library methods known in the art, including but not limited to, biological libraries, spatially addressable parallel solid phase or solution phase libraries, synthetic library methods requiring deconvolution, the one-bead one-compound library method, and synthetic library methods using affinity chromatography selection. The biological library approach is limited to polypeptide libraries, while the other four approaches are applicable to polypeptide, non-peptide oligomer, or small molecule libraries of compounds. [For review see Lam, 1997, (151)].

Methods for the synthesis of molecular libraries are well known in the art [see, for example, DeWitt et al., 1993, (152); Erb et al., 1994, (153); Zuckermann et al., 1994, (154); Cho et al., 1993, (155); Carell et al., 1994, (156) and Gallop et al., 1994, (157). Libraries of compounds can be presented in solution [see, e.g., Houghten, 1992, (158)], or on beads [Lam, 1991, (159)], DNA-chips [Fodor, 1993, (160)], bacteria or spores (Ladner, U.S. Patent 5,223,409), plasmids [Cull et al., 1992, (161)], or phage [Scott & Smith, 1990, (162); Devlin, 1990, (163); Cwirla et al., 1990, (164); Felici, 1991, (165)].

High Throughput Screening

Test compounds can be screened for the ability to bind to „BREAST CANCER GENE“ polypeptides or polynucleotides or to affect „BREAST CANCER GENE“ activity or „BREAST CANCER GENE“ expression using high throughput screening. Using high throughput screening, many discrete compounds can be tested in parallel so that large numbers of test compounds can be quickly screened. The most widely established techniques utilize 96-well, 384-well or 1536-well microtiter plates. The wells of the microtiter plates typically require assay volumes that range from 5 to 500 μ l. In addition to the plates, many instruments, materials, pipettors, robotics, plate washers, and plate readers are commercially available to fit the microwell formats.

Alternatively, free format assays, or assays that have no physical barrier between samples, can be used. For example, an assay using pigment cells (melanocytes) in a simple homogeneous assay for combinatorial peptide libraries is described by Jayawickreme et al., (166). The cells are placed under agarose in culture dishes, then beads that carry combinatorial compounds are placed on the surface of the agarose. The combinatorial compounds are partially released the compounds from the beads. Active compounds can be visualised as dark pigment areas because, as the compounds diffuse locally into the gel matrix, the active compounds cause the cells to change colors.

Another example of a free format assay is described by Chelsky, (167). Chelsky placed a simple homogenous enzyme assay for carbonic anhydrase inside an agarose gel such that the enzyme in the gel would cause a color change throughout the gel. Thereafter, beads carrying combinatorial compounds via a photolinker were placed inside the gel and the compounds were partially released by UV light. Compounds that inhibited the enzyme were observed as local zones of inhibition having less color change.

In another example, combinatorial libraries were screened for compounds that had cytotoxic effects on cancer cells growing in agar [Salmon et al., 1996, (168)].

Another high throughput screening method is described in Beutel et al., U.S. Patent 5,976,813. In this method, test samples are placed in a porous matrix. One or more assay components are then placed within, on top of, or at the bottom of a matrix such as a gel, a plastic sheet, a filter, or other form of easily manipulated solid support. When samples are introduced to the porous matrix they diffuse sufficiently slowly, such that the assays can be performed without the test samples running together.

Binding Assays

For binding assays, the test compound is preferably a small molecule which binds to and occupies, for example, the ATP/GTP binding site of the enzyme or the active site of a „BREAST CANCER GENE“ polypeptide, such that normal biological activity is prevented. Examples of such small molecules include, but are not limited to, small peptides or peptide-like molecules.

In binding assays, either the test compound or a „BREAST CANCER GENE“ polypeptide can comprise a detectable label, such as a fluorescent, radioisotopic, chemiluminescent, or enzymatic label, such as horseradish peroxidase, alkaline phosphatase, or luciferase. Detection of a test compound which is bound to a „BREAST CANCER GENE“ polypeptide can then be accomplished, for example, by direct counting of radioemission, by scintillation counting, or by determining conversion of an appropriate substrate to a detectable product.

Alternatively, binding of a test compound to a „BREAST CANCER GENE“ polypeptide can be determined without labeling either of the interactants. For example, a microphysiometer can be used to detect binding of a test compound with a „BREAST CANCER GENE“ polypeptide. A microphysiometer (e.g., CytosensorJ) is an analytical instrument that measures the rate at which a cell acidifies its environment using a light-addressable potentiometric sensor (LAPS). Changes in this acidification rate can be used as an indicator of the interaction between a test compound and a „BREAST CANCER GENE“ polypeptide [McConnell et al., 1992, (169)].

Determining the ability of a test compound to bind to a „BREAST CANCER GENE“ polypeptide also can be accomplished using a technology such as real-time Bimolecular Interaction Analysis (BIA) [Sjolander & Urbaniczky, 1991, (170), and Szabo et al., 1995, (171)]. BIA is a technology for studying biospecific interactions in real time, without labeling any of the interactants (e.g., BIAcore™). Changes in the optical phenomenon surface plasmon resonance (SPR) can be used as an indication of real-time reactions between biological molecules.

In yet another aspect of the invention, a „BREAST CANCER GENE“ polypeptide can be used as a "bait protein" in a two-hybrid assay or three-hybrid assay [see, e.g., U.S. Patent 5,283,317; Zervos et al., 1993, (172); Madura et al., 1993, (173); Bartel et al., 1993, (174); Iwabuchi et al., 1993, (175) and Brent WO 94/10300], to identify other proteins which bind to or interact with the „BREAST CANCER GENE“ polypeptide and modulate its activity.

The two-hybrid system is based on the modular nature of most transcription factors, which consist of separable DNA-binding and activation domains. Briefly, the assay utilizes two different DNA constructs. For example, in one construct, polynucleotide encoding a „BREAST CANCER GENE“

polypeptide can be fused to a polynucleotide encoding the DNA binding domain of a known transcription factor (e.g., GAL4). In the other construct a DNA sequence that encodes an unidentified protein ("prey" or "sample") can be fused to a polynucleotide that codes for the activation domain of the known transcription factor. If the "bait" and the "prey" proteins are able to interact in vivo to form a protein-dependent complex, the DNA-binding and activation domains of the transcription factor are brought into close proximity. This proximity allows transcription of a reporter gene (e.g., LacZ), which is operably linked to a transcriptional regulatory site responsive to the transcription factor. Expression of the reporter gene can be detected, and cell colonies containing the functional transcription factor can be isolated and used to obtain the DNA sequence encoding the protein which interacts with the „BREAST CANCER GENE“ polypeptide.

It may be desirable to immobilize either a „BREAST CANCER GENE“ polypeptide (or polynucleotide) or the test compound to facilitate separation of bound from unbound forms of one or both of the interactants, as well as to accommodate automation of the assay. Thus, either a „BREAST CANCER GENE“ polypeptide (or polynucleotide) or the test compound can be bound to a solid support. Suitable solid supports include, but are not limited to, glass or plastic slides, tissue culture plates, microtiter wells, tubes, silicon chips, or particles such as beads (including, but not limited to, latex, polystyrene, or glass beads). Any method known in the art can be used to attach a „BREAST CANCER GENE“ polypeptide (or polynucleotide) or test compound to a solid support, including use of covalent and non-covalent linkages, passive absorption, or pairs of binding moieties attached respectively to the polypeptide (or polynucleotide) or test compound and the solid support. Test compounds are preferably bound to the solid support in an array, so that the location of individual test compounds can be tracked. Binding of a test compound to a „BREAST CANCER GENE“ polypeptide (or polynucleotide) can be accomplished in any vessel suitable for containing the reactants. Examples of such vessels include microtiter plates, test tubes, and microcentrifuge tubes.

In one embodiment, a „BREAST CANCER GENE“ polypeptide is a fusion protein comprising a domain that allows the „BREAST CANCER GENE“ polypeptide to be bound to a solid support. For example, glutathione S-transferase fusion proteins can be adsorbed onto glutathione sepharose beads (Sigma Chemical, St. Louis, Mo.) or glutathione derivatized microtiter plates, which are then combined with the test compound or the test compound and the nonadsorbed „BREAST CANCER GENE“ polypeptide; the mixture is then incubated under conditions conducive to complex formation (e.g., at physiological conditions for salt and pH). Following incubation, the beads or microtiter plate wells are washed to remove any unbound components. Binding of the

interactants can be determined either directly or indirectly, as described above. Alternatively, complexes can be dissociated from the solid support before binding is determined.

Other techniques for immobilising proteins or polynucleotides on a solid support also can be used in the screening assays of the invention. For example, either a „BREAST CANCER GENE“ polypeptide (or polynucleotide) or a test compound can be immobilized utilizing conjugation of biotin and streptavidin. Biotinylated „BREAST CANCER GENE“ polypeptides (or polynucleotides) or test compounds can be prepared from biotin NHS (N-hydroxysuccinimide) using techniques well known in the art (e.g., biotinylation kit, Pierce Chemicals, Rockford, Ill.) and immobilized in the wells of streptavidin-coated 96 well plates (Pierce Chemical). Alternatively, antibodies which specifically bind to a „BREAST CANCER GENE“ polypeptide, polynucleotide, or a test compound, but which do not interfere with a desired binding site, such as the ATP/GTP binding site or the active site of the „BREAST CANCER GENE“ polypeptide, can be derivatised to the wells of the plate. Unbound target or protein can be trapped in the wells by antibody conjugation.

Methods for detecting such complexes, in addition to those described above for the GST-immobilized complexes, include immunodetection of complexes using antibodies which specifically bind to a „BREAST CANCER GENE“ polypeptide or test compound, enzyme-linked assays which rely on detecting an activity of a „BREAST CANCER GENE“ polypeptide, and SDS gel electrophoresis under non-reducing conditions.

Screening for test compounds which bind to a „BREAST CANCER GENE“ polypeptide or polynucleotide also can be carried out in an intact cell. Any cell which comprises a „BREAST CANCER GENE“ polypeptide or polynucleotide can be used in a cell-based assay system. A „BREAST CANCER GENE“ polynucleotide can be naturally occurring in the cell or can be introduced using techniques such as those described above. Binding of the test compound to a „BREAST CANCER GENE“ polypeptide or polynucleotide is determined as described above.

Modulation of Gene Expression

In another embodiment, test compounds which increase or decrease „BREAST CANCER GENE“ expression are identified. A „BREAST CANCER GENE“ polynucleotide is contacted with a test compound, and the expression of an RNA or polypeptide product of the „BREAST CANCER GENE“ polynucleotide is determined. The level of expression of appropriate mRNA or polypeptide in the presence of the test compound is compared to the level of expression of mRNA or polypeptide in the absence of the test compound. The test compound can then be identified as a

modulator of expression based on this comparison. For example, when expression of mRNA or polypeptide is greater in the presence of the test compound than in its absence, the test compound is identified as a stimulator or enhancer of the mRNA or polypeptide expression. Alternatively, when expression of the mRNA or polypeptide is less in the presence of the test compound than in its absence, the test compound is identified as an inhibitor of the mRNA or polypeptide expression.

The level of „BREAST CANCER GENE“ mRNA or polypeptide expression in the cells can be determined by methods well known in the art for detecting mRNA or polypeptide. Either qualitative or quantitative methods can be used. The presence of polypeptide products of a „BREAST CANCER GENE“ polynucleotide can be determined, for example, using a variety of techniques known in the art, including immunochemical methods such as radioimmunoassay, Western blotting, and immunohistochemistry. Alternatively, polypeptide synthesis can be determined in vivo, in a cell culture, or in an in vitro translation system by detecting incorporation of labeled amino acids into a „BREAST CANCER GENE“ polypeptide.

Such screening can be carried out either in a cell-free assay system or in an intact cell. Any cell which expresses a „BREAST CANCER GENE“ polynucleotide can be used in a cell-based assay system. A „BREAST CANCER GENE“ polynucleotide can be naturally occurring in the cell or can be introduced using techniques such as those described above. Either a primary culture or an established cell line, such as CHO or human embryonic kidney 293 cells, can be used.

Therapeutic Indications and Methods

Therapies for treatment of breast cancer primarily relied upon effective chemotherapeutic drugs for intervention on the cell proliferation, cell growth or angiogenesis. The advent of genomic driven molecular target identification has opened up the possibility of identifying new breast cancer-specific targets for therapeutic intervention that will provide safer, more effective treatments for malignant neoplasia patients and breast cancer patients in particular. Thus, newly discovered breast cancer-associated genes and their products can be used as tools to develop innovative therapies. The identification of the Her2/neu receptor kinase presents exciting new opportunities for treatment of a certain subset of tumor patients as described before. Genes playing important roles in any of the physiological processes outlined above can be characterized as breast cancer targets. Genes or gene fragments identified through genomics can readily be expressed in one or more heterologous expression systems to produce functional recombinant proteins. These proteins are characterized in vitro for their biochemical properties and then used as tools in high-throughput molecular screening programs to identify chemical modulators of their biochemical

activities. Modulators of target gene expression or protein activity can be identified in this manner and subsequently tested in cellular and in vivo disease models for therapeutic activity. Optimization of lead compounds with iterative testing in biological models and detailed pharmacokinetic and toxicological analyses form the basis for drug development and subsequent testing in humans.

This invention further pertains to the use of novel agents identified by the screening assay described above. Accordingly, it is within the scope of this invention to use a test compound identified as described herein in an appropriate animal model. For example, an agent identified as described herein (e.g., a modulating agent, an antisense polynucleotide molecule, a specific antibody, ribozyme, or a human „BREAST CANCER GENE“ polypeptide binding molecule) can be used in an animal model to determine the efficacy, toxicity, or side effects of treatment with such an agent. Alternatively, an agent identified as described herein can be used in an animal model to determine the mechanism of action of such an agent. Furthermore, this invention pertains to uses of novel agents identified by the above described screening assays for treatments as described herein.

A reagent which affects human „BREAST CANCER GENE“ activity can be administered to a human cell, either in vitro or in vivo, to reduce or increase human „BREAST CANCER GENE“ activity. The reagent preferably binds to an expression product of a human „BREAST CANCER GENE“. If the expression product is a protein, the reagent is preferably an antibody. For treatment of human cells ex vivo, an antibody can be added to a preparation of stem cells which have been removed from the body. The cells can then be replaced in the same or another human body, with or without clonal propagation, as is known in the art.

In one embodiment, the reagent is delivered using a liposome. Preferably, the liposome is stable in the animal into which it has been administered for at least about 30 minutes, more preferably for at least about 1 hour, and even more preferably for at least about 24 hours. A liposome comprises a lipid composition that is capable of targeting a reagent, particularly a polynucleotide, to a particular site in an animal, such as a human. Preferably, the lipid composition of the liposome is capable of targeting to a specific organ of an animal, such as the lung, liver, spleen, heart brain, lymph nodes, and skin.

A liposome useful in the present invention comprises a lipid composition that is capable of fusing with the plasma membrane of the targeted cell to deliver its contents to the cell. Preferably, the transfection efficiency of a liposome is about 0.5 µg of DNA per 16 nmol of liposome delivered to about 10⁶ cells, more preferably about 1.0 µg of DNA per 16 nmol of liposome delivered to about

10^6 cells, and even more preferably about 2.0 μg of DNA per 16 nmol of liposome delivered to about 10^6 cells. Preferably, a liposome is between about 100 and 500 nm, more preferably between about 150 and 450 nm, and even more preferably between about 200 and 400 nm in diameter.

5 Suitable liposomes for use in the present invention include those liposomes usually used in, for example, gene delivery methods known to those of skill in the art. More preferred liposomes include liposomes having a polycationic lipid composition and/or liposomes having a cholesterol backbone conjugated to polyethylene glycol. Optionally, a liposome comprises a compound capable of targeting the liposome to a particular cell type, such as a cell-specific ligand exposed on the outer surface of the liposome.

10 Complexing a liposome with a reagent such as an antisense oligonucleotide or ribozyme can be achieved using methods which are standard in the art (see, for example, U.S. Patent 5,705,151). Preferably, from about 0.1 μg to about 10 μg of polynucleotide is combined with about 8 nmol of liposomes, more preferably from about 0.5 μg to about 5 μg of polynucleotides are combined with about 8 nmol liposomes, and even more preferably about 1.0 μg of polynucleotides is combined
15 with about 8 nmol liposomes.

In another embodiment, antibodies can be delivered to specific tissues in vivo using receptor-mediated targeted delivery. Receptor-mediated DNA delivery techniques are taught in, for example, Findeis et al., 1993, (176); Chiou et al., 1994, (177); Wu & Wu, 1988, (178); Wu et al., 1994, (179); Zenke et al., 1990, (180); Wu et al., 1991, (181).

20 Determination of a Therapeutically Effective Dose

The determination of a therapeutically effective dose is well within the capability of those skilled in the art. A therapeutically effective dose refers to that amount of active ingredient which increases or decreases human „BREAST CANCER GENE“ activity relative to the human „BREAST CANCER GENE“ activity which occurs in the absence of the therapeutically effective
25 dose.

For any compound, the therapeutically effective dose can be estimated initially either in cell culture assays or in animal models, usually mice, rabbits, dogs, or pigs. The animal model also can be used to determine the appropriate concentration range and route of administration. Such information can then be used to determine useful doses and routes for administration in humans.

30 Therapeutic efficacy and toxicity, e.g., ED_{50} (the dose therapeutically effective in 50% of the population) and LD_{50} (the dose lethal to 50% of the population), can be determined by standard

pharmaceutical procedures in cell cultures or experimental animals. The dose ratio of toxic to therapeutic effects is the therapeutic index, and it can be expressed as the ratio, LD_{50}/ED_{50} .

Pharmaceutical compositions which exhibit large therapeutic indices are preferred. The data obtained from cell culture assays and animal studies is used in formulating a range of dosage for human use. The dosage contained in such compositions is preferably within a range of circulating concentrations that include the ED_{50} with little or no toxicity. The dosage varies within this range depending upon the dosage form employed, sensitivity of the patient, and the route of administration.

The exact dosage will be determined by the practitioner, in light of factors related to the subject that requires treatment. Dosage and administration are adjusted to provide sufficient levels of the active ingredient or to maintain the desired effect. Factors which can be taken into account include the severity of the disease state, general health of the subject, age, weight, and gender of the subject, diet, time and frequency of administration, drug combination(s), reaction sensitivities, and tolerance/response to therapy. Long-acting pharmaceutical compositions can be administered every 3 to 4 days, every week, or once every two weeks depending on the half-life and clearance rate of the particular formulation.

Normal dosage amounts can vary from 0.1 to 100,000 micrograms, up to a total dose of about 1 g, depending upon the route of administration. Guidance as to particular dosages and methods of delivery is provided in the literature and generally available to practitioners in the art. Those skilled in the art will employ different formulations for nucleotides than for proteins or their inhibitors. Similarly, delivery of polynucleotides or polypeptides will be specific to particular cells, conditions, locations, etc.

If the reagent is a single-chain antibody, polynucleotides encoding the antibody can be constructed and introduced into a cell either ex vivo or in vivo using well-established techniques including, but not limited to, transferrin-polycation-mediated DNA transfer, transfection with naked or encapsulated nucleic acids, liposome-mediated cellular fusion, intracellular transportation of DNA-coated latex beads, protoplast fusion, viral infection, electroporation, a gene gun, and DEAE- or calcium phosphate-mediated transfection.

Effective in vivo dosages of an antibody are in the range of about 5 μ g to about 50 μ g/kg, about 50 μ g to about 5 mg/kg, about 100 μ g to about 500 μ g/kg of patient body weight, and about 200 to about 250 μ g/kg of patient body weight. For administration of polynucleotides encoding single-chain antibodies, effective in vivo dosages are in the range of about 100 ng to about 200 ng,

500 ng to about 50 mg, about 1 µg to about 2 mg, about 5 µg to about 500 µg, and about 20 µg to about 100 µg of DNA.

If the expression product is mRNA, the reagent is preferably an antisense oligonucleotide or a ribozyme. Polynucleotides which express antisense oligonucleotides or ribozymes can be introduced into cells by a variety of methods, as described above.

Preferably, a reagent reduces expression of a „BREAST CANCER GENE“ gene or the activity of a „BREAST CANCER GENE“ polypeptide by at least about 10, preferably about 50, more preferably about 75, 90, or 100% relative to the absence of the reagent. The effectiveness of the mechanism chosen to decrease the level of expression of a „BREAST CANCER GENE“ gene or the activity of a „BREAST CANCER GENE“ polypeptide can be assessed using methods well known in the art, such as hybridization of nucleotide probes to „BREAST CANCER GENE“ specific mRNA, quantitative RT-PCR, immunologic detection of a „BREAST CANCER GENE“ polypeptide, or measurement of „BREAST CANCER GENE“ activity.

In any of the embodiments described above, any of the pharmaceutical compositions of the invention can be administered in combination with other appropriate therapeutic agents. Selection of the appropriate agents for use in combination therapy can be made by one of ordinary skill in the art, according to conventional pharmaceutical principles. The combination of therapeutic agents can act synergistically to effect the treatment or prevention of the various disorders described above. Using this approach, one may be able to achieve therapeutic efficacy with lower dosages of each agent, thus reducing the potential for adverse side effects.

Any of the therapeutic methods described above can be applied to any subject in need of such therapy, including, for example, birds and mammals such as dogs, cats, cows, pigs, sheep, goats, horses, rabbits, monkeys, and most preferably, humans.

All patents and patent applications cited in this disclosure are expressly incorporated herein by reference. The above disclosure generally describes the present invention. A more complete understanding can be obtained by reference to the following specific examples which are provided for purposes of illustration only and are not intended to limit the scope of the invention.

Pharmaceutical Compositions

The invention also provides pharmaceutical compositions which can be administered to a patient to achieve a therapeutic effect. Pharmaceutical compositions of the invention can comprise, for example, a „BREAST CANCER GENE“ polypeptide, „BREAST CANCER GENE“ polynucleo-

5 tide, ribozymes or antisense oligonucleotides, antibodies which specifically bind to a „BREAS
CANCER GENE“ polypeptide, or mimetics, agonists, antagonists, or inhibitors of a „BREAS
CANCER GENE“ polypeptide activity. The compositions can be administered alone or in
combination with at least one other agent, such as stabilizing compound, which can be
administered in any sterile, biocompatible pharmaceutical carrier, including, but not limited to
saline, buffered saline, dextrose, and water. The compositions can be administered to a patient
alone, or in combination with other agents, drugs or hormones.

10 In addition to the active ingredients, these pharmaceutical compositions can contain suitable
pharmaceutically acceptable carriers comprising excipients and auxiliaries which facilitate
processing of the active compounds into preparations which can be used pharmaceutically.
Pharmaceutical compositions of the invention can be administered by any number of routes,
including, but not limited to, oral, intravenous, intramuscular, intraarterial, intramedullary,
intrathecal, intraventricular, transdermal, subcutaneous, intraperitoneal, intranasal, parenteral,
topical, sublingual, or rectal means. Pharmaceutical compositions for oral administration can be
15 formulated using pharmaceutically acceptable carriers well known in the art in dosages suitable for
oral administration. Such carriers enable the pharmaceutical compositions to be formulated as
tablets, pills, dragees, capsules, liquids, gels, syrups, slurries, suspensions, and the like, for
ingestion by the patient.

20 Pharmaceutical preparations for oral use can be obtained through combination of active
compounds with solid excipient, optionally grinding a resulting mixture, and processing the
mixture of granules, after adding suitable auxiliaries, if desired, to obtain tablets or dragee cores.
Suitable excipients are carbohydrate or protein fillers, such as sugars, including lactose, sucrose,
mannitol, or sorbitol; starch from corn, wheat, rice, potato, or other plants; cellulose, such as
methyl cellulose, hydroxypropylmethylcellulose, or sodium carboxymethylcellulose; gums
25 including arabic and tragacanth; and proteins such as gelatin and collagen. If desired,
disintegrating or solubilizing agents can be added, such as the cross-linked polyvinyl pyrrolidone,
agar, alginic acid, or a salt thereof, such as sodium alginate.

30 Dragee cores can be used in conjunction with suitable coatings, such as concentrated sugar
solutions, which also can contain gum arabic, talc, polyvinylpyrrolidone, carbopol gel,
polyethylene glycol, and/or titanium dioxide, lacquer solutions, and suitable organic solvents or
solvent mixtures. Dyestuffs or pigments can be added to the tablets or dragee coatings for product
identification or to characterize the quantity of active compound, i.e., dosage.

Pharmaceutical preparations which can be used orally include push-fit capsules made of gelatin, as well as soft, sealed capsules made of gelatin and a coating, such as glycerol or sorbitol. Push-fit capsules can contain active ingredients mixed with a filler or binders, such as lactose or starches, lubricants, such as talc or magnesium stearate, and, optionally, stabilizers. In soft capsules, the active compounds can be dissolved or suspended in suitable liquids, such as fatty oils, liquid, or liquid polyethylene glycol with or without stabilizers.

Pharmaceutical formulations suitable for parenteral administration can be formulated in aqueous solutions, preferably in physiologically compatible buffers such as Hanks' solution, Ringer's solution, or physiologically buffered saline. Aqueous injection suspensions can contain substances which increase the viscosity of the suspension, such as sodium carboxymethyl cellulose, sorbitol, or dextran. Additionally, suspensions of the active compounds can be prepared as appropriate oily injection suspensions. Suitable lipophilic solvents or vehicles include fatty oils such as sesame oil or synthetic fatty acid esters, such as ethyl oleate or triglycerides, or liposomes. Non-lipid polycationic amino polymers also can be used for delivery. Optionally, the suspension also can contain suitable stabilizers or agents which increase the solubility of the compounds to allow for the preparation of highly concentrated solutions. For topical or nasal administration, penetrants appropriate to the particular barrier to be permeated are used in the formulation. Such penetrants are generally known in the art.

The pharmaceutical compositions of the present invention can be manufactured in a manner that is known in the art, e.g., by means of conventional mixing, dissolving, granulating, dragee making, levigating, emulsifying, encapsulating, entrapping, or lyophilizing processes. The pharmaceutical composition can be provided as a salt and can be formed with many acids, including but not limited to, hydrochloric, sulfuric, acetic, lactic, tartaric, malic, succinic, etc. Salts tend to be more soluble in aqueous or other protonic solvents than are the corresponding free base forms. In other cases, the preferred preparation can be a lyophilized powder which can contain any or all of the following: 150 mM histidine, 0.1%2% sucrose, and 27% mannitol, at a pH range of 4.5 to 5.5, that is combined with buffer prior to use.

Further details on techniques for formulation and administration can be found in the latest edition of REMINGTON'S PHARMACEUTICAL SCIENCES (182). After pharmaceutical compositions have been prepared, they can be placed in an appropriate container and labeled for treatment of an indicated condition. Such labeling would include amount, frequency, and method of administration.

MATERIAL AND METHODS

One strategy for identifying genes that are involved in breast cancer is to detect genes that are expressed differentially under conditions associated with the disease versus non-disease conditions. The sub-sections below describe a number of experimental systems which may be used to detect such differentially expressed genes. In general, these experimental systems include at least one experimental condition in which subjects or samples are treated in a manner associated with breast cancer, in addition to at least one experimental control condition lacking such disease-associated treatment. Differentially expressed genes are detected, as described below, by comparing the pattern of gene expression between the experimental and control conditions.

Once a particular gene has been identified through the use of one such experiment, its expression pattern may be further characterized by studying its expression in a different experiment and the findings may be validated by an independent technique. Such use of multiple experiments may be useful in distinguishing the roles and relative importance of particular genes in breast cancer. A combined approach, comparing gene expression pattern in cells derived from breast cancer patients to those of *in vitro* cell culture models can give substantial hints on the pathways involved in development and/or progression of breast cancer.

Among the experiments which may be utilized for the identification of differentially expressed genes involved in malignant neoplasia and breast cancer, for example, are experiments designed to analyze those genes which are involved in signal transduction. Such experiments may serve to identify genes involved in the proliferation of cells.

Below are methods described for the identification of genes which are involved in breast cancer. Such represent genes which are differentially expressed in breast cancer conditions relative to their expression in normal, or non-breast cancer conditions or upon experimental manipulation based on clinical observations. Such differentially expressed genes represent "target" and/or "marker" genes. Methods for the further characterization of such differentially expressed genes, and for their identification as target and/or marker genes, are presented below.

Alternatively, a differentially expressed gene may have its expression modulated, i.e., quantitatively increased or decreased, in normal versus breast cancer states, or under control versus experimental conditions. The degree to which expression differs in normal versus breast cancer or control versus experimental states need only be large enough to be visualized via standard characterization techniques, such as, for example, the differential display technique described below. Other such standard characterization techniques by which expression differences

may be visualized include but are not limited to quantitative RT-PCR and Northern analyses, which are well known to those of skill in the art.

As part of this invention, a method is described by way of illustration and not by limitation, displaying at least some of the below mentioned aspects:

- 5 1. A method for the prediction, diagnosis or prognosis of malignant neoplasia by the detection of at least 2 markers characterized in that the markers are genes and fragments thereof or genomic nucleic acid sequences that are located on one chromosomal region which is altered in malignant neoplasia.
- 10 2. A method for the prediction, diagnosis or prognosis of malignant neoplasia by the detection of at least 2 markers characterized in that the markers are:
 - a) genes that are located on one or more chromosomal region(s) which is/are altered in malignant neoplasia; and
 - b)
 - 15 i) receptor and ligand; or
 - ii) members of the same signal transduction pathway; or
 - iii) members of synergistic signal transduction pathways; or
 - iv) members of antagonistic signal transduction pathways; or
 - v) transcription factor and transcription factor binding site.
- 20 3. The method of aspect 1 or 2 wherein the malignant neoplasia is breast cancer, ovarian cancer, gastric cancer, colon cancer, esophageal cancer, mesenchymal cancer, bladder cancer or non-small cell lung cancer.
4. The method of aspect 1 or 2 wherein at least one chromosomal region is defined as the cytogenetic region: 1p13, 1q32, 3p21-p24, 5p13-p14, 8q23-q24, 11q13, 12q13, 17q12-q24 or 20q13.
- 25 5. The method of aspect 1 or 2 wherein at least chromosomal region is defined as the cytogenetic region 17q11.2-21.3 and the malignant neoplasia is breast cancer, ovarian cancer, gastric cancer, colon cancer, esophageal cancer, mesenchymal cancer, bladder cancer or non-small cell lung cancer.

6. The method of aspect 1 or 2 wherein at least one chromosomal region is defined as the cytogenetic region 3p21-24 and the malignant neoplasia is breast cancer, ovarian cancer, gastric cancer, colon cancer, esophageal cancer, mesenchymal cancer, bladder cancer or non-small cell lung cancer.
- 5 7. The method of aspect 1 or 2 wherein at least one chromosomal region is defined as the cytogenetic region 12q13 and the malignant neoplasia is breast cancer, ovarian cancer, gastric cancer, colon cancer, esophageal cancer, mesenchymal cancer, bladder cancer or non-small cell lung cancer.
- 10 8. A method for the prediction, diagnosis or prognosis of malignant neoplasia by the detection of at least one marker whereby the marker is a VNTR, SNP, RFLP or STR characterized in that the marker is located on one chromosomal region which is altered in malignant neoplasia due to amplification and the marker is detected in a cancerous and non-cancerous tissue or biological sample of the same individual.
- 15 9. The method of aspect 8 wherein the marker is selected from the group consisting of the VNTRs:
D17S946, D17S1181, D17S2026, D17S838, D17S250, D17S1818, D17S614, D17S2019, D17S608, D17S1655, D17S2147, D17S754, D17S1814, D17S2007, D17S1246, D17S1979, D17S1984, D17S1984, D17S1867, D17S1788, D17S1836, D17S1787, D17S1660, D17S2154, D17S1955, D17S2098, D17S518, D17S1851, D17S4358, D17S964, D17S1091, D17S1179, D17S2160, D17S1230, D17S1338, D17S2011, D17S1237, D17S2038, D17S2091, D17S649, D17S1190 and M87506.
- 20 10. The method of aspect 8 wherein the marker is selected from the group consisting of the SNPs:
rs2230698, rs2230700, rs1058808, rs1801200, rs903506, rs2313170, rs1136201, rs2934968, rs2172826, rs1810132, rs1801201, rs2230702, rs2230701, rs1126503, rs3471, rs13695, rs471692, rs558068, rs1064288, rs1061692, rs520630, rs782774, rs565121, rs2586112, rs532299, rs2732786, rs1804539, rs1804538, rs1804537, rs1141364, rs12231, rs1132259, rs1132257, rs1132256, rs1132255, rs1132254, rs1132252, rs1132268 and rs1132258
- 25 11. A method for the prediction, diagnosis or prognosis of malignant neoplasia by the detection of at least one marker characterized in that the marker is selected from:
- 30

- 5
- a) a polynucleotide or polynucleotide analog comprising at least one of the sequences of SEQ ID NO: 2 to 6, 8, 9, 11 to 16, 18, 19, 21 to 26, 53 to 75, or 315 to 318 ;
 - b) a polynucleotide or polynucleotide analog which hybridizes under stringent conditions to a polynucleotide specified in (a) and encodes a polypeptide exhibiting the same biological function as specified for the respective sequence in Table 2 or 3
 - c) a polynucleotide or polynucleotide analog the sequence of which deviates from the polynucleotide specified in (a) and (c) due to the generation of the genetic code encoding a polypeptide exhibiting the same biological function as specified for the respective sequence in Table 2 or 3
 - 10 d) a polynucleotide or polynucleotide analog which represents a specific fragment, derivative or allelic variation of a polynucleotide sequence specified in (a) to (d)
 - e) a purified polypeptide encoded by a polynucleotide or polynucleotide analog sequence specified in (a) to (e)
 - 15 f) A purified polypeptide comprising at least one of the sequences of SEQ ID NO: 28 to 32, 34, 35, 37 to 42, 44, 45, 47 to 52, 76 to 98, or 393 to 396;
- are detected.
12. A method for the prediction, diagnosis or prognosis of malignant neoplasia by the detection of at least 2 markers characterized in that at least 2 markers are selected from:
- 20
- a) polynucleotide or polynucleotide analog comprising at least one of the sequences of SEQ ID NO: 1 to 26 or 53 to 75 or 315 to 318;
 - b) a polynucleotide or polynucleotide analog which hybridizes under stringent conditions to a polynucleotide specified in (a) and encodes a polypeptide exhibiting the same biological function as specified for the respective sequence in Table 2 or 3
 - 25 c) a polynucleotide or polynucleotide analog the sequence of which deviates from the polynucleotide specified in (a) and (b) due to the generation of the genetic code encoding a polypeptide exhibiting the same biological function as specified for the respective sequence in Table 2 or 3

- d) a polynucleotide or polynucleotide analog which represents a specific fragment derivative or allelic variation of a polynucleotide sequence specified in (a) to (c)
- e) a purified polypeptide encoded by a polynucleotide sequence or polynucleotide analog specified in (a) to (d)
- 5 f) a purified polypeptide comprising at least one of the sequences of SEQ ID NO: 2 to 52 or 76 to 98 or 393 to 396
- are detected.
13. The method of any of the aspects 1 or 12 wherein the detection method comprises the use of PCR, arrays or beads.
- 10 14. A diagnostic kit comprising instructions for conducting the method of any of aspects 1 to 13.
15. A composition for the prediction, diagnosis or prognosis of malignant neoplasia comprising:
- a) a detection agent for:
- 15 i) any polynucleotide or polynucleotide analog comprising at least one of the sequences of SEQ ID NO: 2 to 6, 8, 9, 11 to 16, 18, 19, 21 to 26, 53 to 75 or 315 to 318,
- 20 ii) any polynucleotide or polynucleotide analog which hybridizes under stringent conditions to a polynucleotide specified in (a) encoding a polypeptide exhibiting the same biological function as specified for the respective sequence in Table 2 or 3
- 25 iii) a polynucleotide or polynucleotide analog the sequence of which deviates from the polynucleotide specified in (a) and (b) due to the generation of the genetic code encoding a polypeptide exhibiting the same biological function as specified for the respective sequence in Table 2 or 3
- iv) a polynucleotide or polynucleotide analog which represents a specific fragment, derivative or allelic variation of a polynucleotide sequence specified in (a) to (c)

- v) a polypeptide encoded by a polynucleotide or polynucleotide analog sequence specified in (a) to (d);
- vi) a polypeptide comprising at least one of the sequences of SEQ ID NO: 28 to 32, 34, 35, 37 to 42, 44, 45, 47 to 52, 76 to 98, or 393 to 396.

5 or

b) at least 2 detection agents for at least 2 markers selected from:

- i) any polynucleotide comprising at least one of the sequences of SEQ ID NO: 1 to 26 or 53 to 75 or 315 to 318;
- 10 ii) any polynucleotide which hybridizes under stringent conditions to a polynucleotide specified in (a) encoding a polypeptide exhibiting the same biological function as specified for the respective sequence in Table 2 or 3
- 15 iii) a polynucleotide the sequence of which deviates from the polynucleotide specified in (a) and (b) due to the generation of the genetic code encoding a polypeptide exhibiting the same biological function as specified for the respective sequence in Table 2 or 3
- iv) a polynucleotide which represents a specific fragment, derivative or allelic variation of a polynucleotide sequence specified in (a) to (c)
- v) a polypeptide encoded by a polynucleotide sequence specified in (a) to (d);
- 20 vi) a polypeptide comprising at least one of the sequences of SEQ ID NO: to 52 or 76 to 98 or 393 to 396.

16. An array comprising a plurality of polynucleotides or polynucleotide analogs wherein each of the polynucleotides is selected from:

- a) a polynucleotide or polynucleotide analog comprising at least one of the sequences of SEQ ID NO: 1 to 26 or 53 to 75 or 315 to 318;
- 25 b) a polynucleotide or polynucleotide analog which hybridizes under stringent conditions to a polynucleotide specified in (a) encoding a polypeptide exhibiting the same biological function as specified for the respective sequence in Table 2 or 3

- c) a polynucleotide or polynucleotide analog the sequence of which deviates from the polynucleotide specified in (a) and (b) due to the generation of the genetic code encoding a polypeptide exhibiting the same biological function as specified for the respective sequence in Table 2 or 3
- 5 d) a polynucleotide or polynucleotide analog which represents a specific fragment, derivative or allelic variation of a polynucleotide sequence specified in (a) to (c) attached to a solid support.
17. A method of screening for agents which regulate the activity of a polypeptide encoded by a polynucleotide or polynucleotide analog selected from the group consisting of:
- 10 a) a polynucleotide or polynucleotide analog comprising at least one of the sequences of SEQ ID NO: 2 to 6, 8, 9, 11 to 16, 18, 19, 21 to 26, 53 to 75 or 315 to 318;
- b) a polynucleotide or polynucleotide analog which hybridizes under stringent conditions to a polynucleotide specified in (a) encoding a polypeptide exhibiting the same biological function as specified for the respective sequence in Table 2 or 3
- 15 c) a polynucleotide or polynucleotide analog the sequence of which deviates from the polynucleotide specified in (a) and (b) due to the generation of the genetic code encoding a polypeptide exhibiting the same biological function as specified for the respective sequence in Table 2 or 3
- 20 d) a polynucleotide or polynucleotide analog which represents a specific fragment, derivative or allelic variation of a polynucleotide sequence specified in (a) to (c); comprising the steps of:
- i) contacting a test compound with at least one polypeptide encoded by a polynucleotide specified in (a) to (d); and
- 25 ii) detecting binding of the test compound to the polypeptide, wherein a test compound which binds to the polypeptide is identified as a potential therapeutic agent for modulating the activity of the polypeptide in order to prevent of treat malignant neoplasia.

18. A method of screening for agents which regulate the activity of a polypeptide encoded by a polynucleotide or polynucleotide analog selected from the group consisting of:
- a) a polynucleotide or polynucleotide analog comprising at least one of the sequences of SEQ ID NO: 2 to 6, 8, 9, 11 to 16, 18, 19, 21 to 26, 53 to 75, or 315 to 318;
 - 5 b) a polynucleotide or polynucleotide analog which hybridizes under stringent conditions to a polynucleotide specified in (a) encoding a polypeptide exhibiting the same biological function as specified for the respective sequence in Table 2 or 3
 - 10 c) a polynucleotide or polynucleotide analog the sequence of which deviates from the polynucleotide specified in (a) and (b) due to the generation of the genetic code encoding a polypeptide exhibiting the same biological function as specified for the respective sequence in Table 2 or 3
 - d) a polynucleotide or polynucleotide analog which represents a specific fragment, derivative or allelic variation of a polynucleotide sequence specified in (a) to (c)
- 15 comprising the steps of:
- i) contacting a test compound with at least one polypeptide encoded by a polynucleotide specified in (a) to (d); and
 - ii) detecting the activity of the polypeptide as specified for the respective sequence in Table 2 or 3, wherein a test compound which increases the activity is identified as a potential preventive or therapeutic agent for increasing the polypeptide activity in malignant neoplasia, and wherein a test compound which decreases the activity of the polypeptide is identified as a potential therapeutic agent for decreasing the polypeptide activity in malignant neoplasia.
- 20
19. A method of screening for agents which regulate the activity of a polynucleotide or polynucleotide analog selected from group consisting of;
- 25
- a) a polynucleotide or polynucleotide analog comprising at least one of the sequences of SEQ ID NO: 2 to 6, 8, 9, 11 to 16, 18, 19, 21 to 26, 53 to 75 or 315 to 318;
 - b) a polynucleotide or polynucleotide analog which hybridizes under stringent conditions to a polynucleotide specified in (a) encoding a polypeptide exhibiting

the same biological function as specified for the respective sequence in Table 2 or 3

- 5
- c) a polynucleotide or polynucleotide analog the sequence of which deviates from the polynucleotide specified in (a) and (b) due to the generation of the genetic code encoding a polypeptide exhibiting the same biological function as specified for the respective sequence in Table 2 or 3
 - d) a polynucleotide or polynucleotide analog which represents a specific fragment, derivative or allelic variation of a polynucleotide sequence specified in (a) to (c)

comprising the steps of:

- 10
- i) contacting a test compound with at least one polynucleotide or polynucleotide analog specified in (a) to (d), and
 - ii) detecting binding of the test compound to the polynucleotide, wherein a test compound which binds to the polynucleotide is identified as a potential preventive or therapeutic agent for regulating the activity of the polynucleotide in malignant
- 15
- neoplasia.

20. Use of

- 20
- a) a polynucleotide or polynucleotide analog comprising at least one of the sequences of SEQ ID NO: 2 to 6, 8, 9, 11 to 16, 18, 19, 21 to 26, 53 to 75 or 315 to 318;
 - b) a polynucleotide which hybridizes under stringent conditions to a polynucleotide or polynucleotide analog specified in (a) encoding a polypeptide exhibiting the same biological function as specified for the respective sequence in Table 2 or 3;
 - c) a polynucleotide or polynucleotide analog the sequence of which deviates from the polynucleotide specified in (a) and (b) due to the generation of the genetic code encoding a polypeptide exhibiting the same biological function as specified for the
- 25
- d) a polynucleotide or polynucleotide analog which represents a specific fragment, derivative or allelic variation of a polynucleotide sequence specified in (a) to (c);
 - e) an antisense molecule targeting specifically one of the polynucleotide sequences specified in (a) to (d);

- f) a purified polypeptide encoded by a polynucleotide or polynucleotide analog sequence specified in (a) to (d)
- g) a purified polypeptide comprising at least one of the sequences of SEQ ID NO: 28 to 32, 34, 35, 37 to 42, 44, 45, 47 to 52, 76 to 98 or 393 to 396;
- 5 h) an antibody capable of binding to one of the polynucleotide specified in (a) to (d) or a polypeptide specified in (f) and (g);
- i) a reagent identified by any of the methods of aspect 17 to 19 that modulates the amount or activity of a polynucleotide sequence specified in (a) to (d) or a polypeptide specified in (f) and (g);
- 10 in the preparation of a composition for the prevention, prediction, diagnosis, prognosis or medicament for the treatment of malignant neoplasia.
21. Use of aspect 20 wherein the disease is breast cancer.
22. A reagent that regulates the activity of a polypeptide selected from the group consisting of:
- 15 a) a polypeptide encoded by any polynucleotide or polynucleotide analog comprising at least one of the sequences of SEQ ID NO: 2 to 6, 8, 9, 11 to 16, 18, 19, 21 to 26, 53 to 75 or 315 to 318;
- 20 b) a polypeptide encoded by any polynucleotide or polynucleotide analog which hybridizes under stringent conditions to any polynucleotide comprising at least one of the sequences of SEQ ID NO: 2 to 6, 8, 9, 11 to 16, 18, 19, 21 to 26, 53 to 75 or 315 to 318 encoding a polypeptide exhibiting the same biological function as specified for the respective sequence in Table 2 or 3
- 25 c) a polypeptide encoded by any polynucleotide or polynucleotide analog the sequence of which deviates from the polynucleotide specified in (a) and (b) due to the generation of the genetic code encoding a polypeptide exhibiting the same biological function as specified for the respective sequence in Table 2 or 3
- d) a polypeptide encoded by any polynucleotide or polynucleotide analog which represents a specific fragment, derivative or allelic variation of a polynucleotide sequence specified in (a) to (c) encoding a polypeptide exhibiting the same biological function as specified for the respective sequence in Table 2 or 3

- e) or a polypeptide comprising at least one of the sequences of SEQ ID NO: 28 to 34, 35, 37 to 42, 44, 45, 47 to 52, 76 to 98 or 393 to 396;

wherein said reagent is identified by the method of any of the aspects 17 to 19.

23. A reagent that regulates the activity of a polynucleotide or polynucleotide analog selected from the group consisting of:

5

- a) a polynucleotide or polynucleotide analog comprising at least one of the sequences of SEQ ID NO: 2 to 6, 8, 9, 11 to 16, 18, 19, 21 to 26, 53 to 75 or 315 to 318;

- b) a polynucleotide or polynucleotide analog which hybridizes under stringent conditions to a polynucleotide specified in (a) encoding a polypeptide exhibiting the same biological function as specified for the respective sequence in Table 2 or 3

10

- c) a polynucleotide or polynucleotide analog the sequence of which deviates from the polynucleotide specified in (a) and (b) due to the generation of the genetic code encoding a polypeptide exhibiting the same biological function as specified for the respective sequence in Table 2 or 3

15

- d) a polynucleotide or polynucleotide analog which represents a specific fragment, derivative or allelic variation of a polynucleotide sequence specified in (a) to (c) encoding a polypeptide exhibiting the same biological function as specified for the respective sequence in Table 2 or 3

20

wherein said reagent is identified by the method of any of the aspects 17 to 19.

24. A pharmaceutical composition, comprising:

- a) an expression vector containing at least one polynucleotide or polynucleotide analog selected from the group consisting of:

25

- i) a polynucleotide or polynucleotide analog comprising at least one of the sequences of SEQ ID NO: 2 to 6, 8, 9, 11 to 16, 18, 19, 21 to 26, 53 to 75 or 315 to 318;

- ii) a polynucleotide or polynucleotide analog which hybridizes under stringent conditions to a polynucleotide specified in (a) encoding a

polypeptide exhibiting the same biological function as specified for the respective sequence in Table 2 or 3

iii) a polynucleotide or polynucleotide analog the sequence of which deviates from the polynucleotide specified in (a) and (b) due to the generation of the genetic code encoding a polypeptide exhibiting the same biological function as specified for the respective sequence in Table 2 or 3

iv) a polynucleotide or polynucleotide analog which represents a specific fragment, derivative or allelic variation of a polynucleotide sequence specified in (a) to (c) encoding a polypeptide exhibiting the same biological function as specified for the respective sequence in Table 2 or 3;

or the reagent of aspect 22 or 23 and a pharmaceutically acceptable carrier.

25. A computer-readable medium comprising:

- a) at least one digitally encoded value representing a level of expression of at least one polynucleotide sequence of SEQ ID NO: 2 to 6, 8, 9, 11 to 16, 18, 19, 21 to 26, 53 to 75 or 315 to 318
- b) at least 2 digitally encoded values representing the levels of expression of at least 2 polynucleotide sequences selected from SEQ ID NO: 1 to 26, 53 to 75 or 315 to 318

in a cell from the a subject at risk for or having malignant neoplasia.

26. A method for the detection of chromosomal alterations characterized in that the relative abundance of individual mRNAs, encoded by genes, located in altered chromosomal regions is detected.

27. A method for the detection of chromosomal alterations characterized in that the copy number of one or more chromosomal region(s) is detected by quantitative PCR.

EXAMPLE 1*Expression profiling**a) Expression profiling utilizing quantitative RT-PCR*

For a detailed analysis of gene expression by quantitative PCR methods, one will utilize primers flanking the genomic region of interest and a fluorescent labeled probe hybridizing in-between. Using the PRISM 7700 Sequence Detection System of PE Applied Biosystems (Perkin Elmer, Foster City, CA, USA) with the technique of a fluorogenic probe, consisting of an oligonucleotide labeled with both a fluorescent reporter dye and a quencher dye, one can perform such expression measurement. Amplification of the probe-specific product causes cleavage of the probe, generating an increase in reporter fluorescence. Primers and probes were selected using the Primer Express software and localized mostly in the 3' region of the coding sequence or in the untranslated region (see Table 5 for primer- and probe- sequences) according to the relative positions of the probe sequence used for the construction of the Affymetrix HG_U95A-E or HGU133A-B DNA-chips. All primer pairs were checked for specificity by conventional PCR reactions. To standardize the amount of sample RNA, GAPDH was selected as a reference, since it was not differentially regulated in the samples analyzed. TaqMan validation experiments were performed showing that the efficiencies of the target and the control amplifications are approximately equal which is a prerequisite for the relative quantification of gene expression by the comparative $\Delta\Delta C_T$ method, known to those with skills in the art.

As well as the technology provided by Perkin Elmer one may use other technique implementations like LightcyclerTM from Roche Inc. or iCycler from Stratagene Inc..

b) Expression profiling utilizing DNA microarrays

Expression profiling can be carried out using the Affymetrix Array Technology. By hybridization of mRNA to such a DNA-array or DNA-Chip, it is possible to identify the expression value of each transcripts due to signal intensity at certain position of the array. Usually these DNA-arrays are produced by spotting of cDNA, oligonucleotides or subcloned DNA fragments. In case of Affymetrix technology app. 400.000 individual oligonucleotide sequences were synthesized on the surface of a silicon wafer at distinct positions. The minimal length of oligomers is 12 nucleotides, preferable 25 nucleotides or full length of the questioned transcript. Expression profiling may also be carried out by hybridization to nylon or nitro-cellulose membrane bound DNA or oligonucleotides. Detection of signals derived from hybridization may be obtained by either colorimetric, fluorescent, electrochemical, electronic, optic or by radioactive readout. Detailed

description of array construction have been mentioned above and in other patents cited. To determine the quantitative and qualitative changes in the chromosomal region to analyze, RNA from tumor tissue which is suspected to contain such genomic alterations has to be compared to RNA extracted from benign tissue (e.g. epithelial breast tissue, or micro dissected ductal tissue) on the basis of expression profiles for the whole transcriptome. With minor modifications, the sample preparation protocol followed the Affymetrix GeneChip Expression Analysis Manual (Santa Clara, CA). Total RNA extraction and isolation from tumor or benign tissues, biopsies, cell isolates or cell containing body fluids can be performed by using TRIzol (Life Technologies, Rockville, MD) and Oligotex mRNA Midi kit (Qiagen, Hilden, Germany), and an ethanol precipitation step should be carried out to bring the concentration to 1 mg/ml. Using 5–10 mg of mRNA to create double stranded cDNA by the SuperScript system (Life Technologies). First strand cDNA synthesis was primed with a T7-(dT24) oligonucleotide. The cDNA can be extracted with phenol/chloroform and precipitated with ethanol to a final concentration of 1mg /ml. From the generated cDNA, cRNA can be synthesized using Enzo's (Enzo Diagnostics Inc., Farmingdale, NY) *in vitro* Transcription Kit. Within the same step the cRNA can be labeled with biotin nucleotides Bio-11-CTP and Bio-16-UTP (Enzo Diagnostics Inc., Farmingdale, NY) . After labeling and cleanup (Qiagen, Hilden (Germany) the cRNA then should be fragmented in an appropriated fragmentation buffer (e.g., 40 mM Tris-Acetate, pH 8.1, 100 mM KOAc, 30 mM MgOAc, for 35 minutes at 94°C). As per the Affymetrix protocol, fragmented cRNA should be hybridized on the HG_U133 arrays A and B, comprising app. 40.000 probed transcripts each, for 24 hours at 60 rpm in a 45°C hybridization oven. After Hybridization step the chip surfaces have to be washed and stained with streptavidin phycoerythrin (SAPE; Molecular Probes, Eugene, OR) in Affymetrix fluidics stations. To amplify staining, a second labeling step can be introduced, which is recommended but not compulsive. Here one should add SAPE solution twice with an antistreptavidin biotinylated antibody. Hybridization to the probe arrays may be detected by fluorometric scanning (Hewlett Packard Gene Array Scanner; Hewlett Packard Corporation, Palo Alto, CA).

After hybridization and scanning, the microarray images can be analyzed for quality control, looking for major chip defects or abnormalities in hybridization signal. Therefor either Affymetrix GeneChip MAS 5.0 Software or other microarray image analysis software can be utilized. Primary data analysis should be carried out by software provided by the manufacturer..

In case of the genes analyses in one embodiment of this invention the primary data have been analyzed by further bioinformatic tools and additional filter criteria. The bioinformatic analysis is described in detail below.

c) Data analysis

According to Affymetrix measurement technique (Affymetrix GeneChip Expression Analysis Manual, Santa Clara, CA) a single gene expression measurement on one chip yields the average difference value and the absolute call. Each chip contains 16–20 oligonucleotide probe pairs per gene or cDNA clone. These probe pairs include perfectly matched sets and mismatched sets, both of which are necessary for the calculation of the average difference, or expression value, a measure of the intensity difference for each probe pair, calculated by subtracting the intensity of the mismatch from the intensity of the perfect match. This takes into consideration variability in hybridization among probe pairs and other hybridization artifacts that could affect the fluorescence intensities. The average difference is a numeric value supposed to represent the expression value of that gene. The absolute call can take the values 'A' (absent), 'M' (marginal), or 'P' (present) and denotes the quality of a single hybridization. We used both the quantitative information given by the average difference and the qualitative information given by the absolute call to identify the genes which are differentially expressed in biological samples from individuals with breast cancer versus biological samples from the normal population. With other algorithms than the Affymetrix one we have obtained different numerical values representing the same expression values and expression differences upon comparison.

The differential expression E in one of the breast cancer groups compared to the normal population is calculated as follows. Given n average difference values d_1, d_2, \dots, d_n in the breast cancer population and m average difference values c_1, c_2, \dots, c_m in the population of normal individuals, it is computed by the equation:

$$E \equiv \exp\left(\frac{1}{m} \sum_{i=1}^m \ln(c_i) - \frac{1}{n} \sum_{i=1}^n \ln(d_i)\right)$$

If $d_j < 50$ or $c_i < 50$ for one or more values of i and j , these particular values c_i and/or d_j are set to an "artificial" expression value of 50. This particular computation of E allows for a correct comparison to TaqMan results.

A gene is called up-regulated in breast cancer versus normal if $E \geq 1.5$ and if the number of absolute calls equal to 'P' in the breast cancer population is greater than $n/2$.

A gene is called down-regulated in breast cancer versus normal if $E \leq -1.5$ and if the number of absolute calls equal to 'P' in the normal population is greater than $m/2$.

The final list of differentially regulated genes consists of all up-regulated and all down-regulated genes in biological samples from individuals with breast cancer versus biological samples from the normal population. Those genes on this list which are interesting for a pharmaceutical application were finally validated by TaqMan. If a good correlation between the expression values/behavior of a transcript could be observed with both techniques, such a gene is listed in Tables 1 to 3.

Since not only the information on differential expression of a single gene within an identified ARCHEON, but also the information on the co-regulation of several members is important for predictive, diagnostic, preventive and therapeutic purposes we have combined expression data with information on the chromosomal position (e.g. golden path) taken from public available databases to develop a picture of the overall transcriptom of a given tumor sample. By this technique not only known or suspected regions of genomes can be inspected but even more valuable, new regions of dysregulation with chromosomal linkage can be identified. This is of value in other types of neoplasia or viral integration and chromosomal rearrangements. By SQL based database searches one can retrieve information on expression, qualitative value of a measurement (denoted by Affymetrix MAS 5.0 Software), expression values derived from other techniques than DNA-chip hybridization and chromosomal linkage.

EXAMPLE 2

Identification of the ARCHEON

a) Identification and localization of genes or gene probes (represented by the so called probe sets on Affymetrix arrays HG-U95A-E or HG-U133A-B) in their chromosomal context and order on the human genome.

For identification of larger chromosomal changes or aberrations, as they have been described in detail above, a sufficient number of genes, transcripts or DNA-fragments is needed. The density of probes covering a chromosomal region is not necessarily limited to the transcribed genes, in case of the use of array based CGH but by utilizing RNA as probe material the density is given by the distance of genes on a chromosome. The DNA-microarrays provided by Affymetrix Inc. Do contain hitherto all transcripts from the known humane genome, which are be represented by 40.000 – 60.000 probe sets. By BLAST mapping and sorting the sequences of these short DNA-oligomers to the public available sequence of the human genome represented by the so called “golden path”, available at the university of California in Santa Cruz or from the NCBI, a chromosomal display of the whole Transcriptome of a tissue specimen evolves. By graphical display of the individual chromosomal regions and color coding of over or under represented

transcripts, compared to a reference transcriptome regions with DNA gains and losses can be identified.

b) Quantification of gene copy numbers by combined IHC and quantitative PCR (PCR karyotyping) or directly by quantitative PCR

5 Usually one to three paraffin-embedded tissue sections that are 5 µm thick are used to obtain genomic DNA from the samples. Tissue sections are stained by colorimetric IHC after deparaffinization to identify regions containing disease associated cells. Stained regions are macrodissected with a scalpel and transferred into a micro-centrifuge tube. The genomic DNA of these isolated tissue sections is extracted using appropriate buffers. The isolated DNA is then used for quantitative PCR with appropriate primers and probes. Optionally the IHC staining can be omitted and the genomic DNA can be directly isolated with or without prior deparaffinization with appropriate buffers. Those who are skilled in the art may vary the conditions and buffers described below to obtain equivalent results.

15 Reagents from DAKO (HercepTest Code No. K 5204) and TaKaRa were used (Biomedicals Cat.: 9091) according to the manufacturer's protocol.

It is convenient to prepare the following reagents prior to staining:

Solution No. 7

Epitope Retrieval Solution (Citrate buffer + antimicrobial agent) (10xconc.)

20 ml ad 200 ml aqua dest. (stable for 1 month at 2-8°C)

20 **Solution No. 8**

Washing-buffer (Tris-HCl + antimicrobial agent) (10 x conc.)

30 ml ad 300 ml distilled water (stable for 1 month at 2-8°C)

Staining solution: DAB

1 ml solution is sufficient for 10 slides. The solution were prepared immediately before usage.:

25 1 ml DAB buffer (Substrate Buffer solution, pH 7.5, containing H₂O₂, stabilizer, enhancers and an antimicrobial agent) + 1 drop (25-3 µl) DAB-Chromogen (3,3'-diaminobenzidine chromogen solution). This solution is stable for up to 5 days at 2-8°C. Precipitated substances do not influence the staining result. Additionally required are: 2 x approx. 100 ml Xylol, 2 x approx. 100 ml Ethanol

100%, 2 x Ethanol 95%, aqua dest. These solution can be used for up to 40 stainings. A water bath is required for the epitope retrieval step.

Staining procedure:

5 All reagents are pre-warmed to room temperature (20-25°C) prior to immunostaining. Likewise all incubations were performed at room temperature. Except the epitope retrieval which is performed in at 95°C water bath. Between the steps excess of liquid is tapped off from the slides with lintless tissue (Kim Wipe).

Deparaffinization

10 Slides are placed in a xylene bath and incubated for 5 minutes. The bath is changed and the step repeated once. Excess of liquid is tapped off and the slides are placed in absolute ethanol for minutes. The bath is changed and the step repeated once. Excess of liquid is tapped off and the slides are placed in 95% ethanol for 3 minutes. The bath is changed and the step repeated once. Excess of liquid is tapped off and the slides are placed in distilled water for a minimum of 30 seconds.

15 Epitope Retrieval

Staining jars are filled with with diluted epitope retrieval solution and preheated in a water bath at 95°C. The deparaffinized sections are immersed into the preheated solution in the staining jars and incubated for 40 minutes at 95°C. The entire jar is removed from the water bath and allowed to cool down at room temperature for 20 minutes. The epitope retrieval solution is decanted, the sections are rinsed in distilled water and finally soaked in wash buffer for 5 minutes.

Peroxidase Blocking:

Excess of buffer is tapped off and the tissue section encircled with a DAKO pen. The specimen is covered with 3 drops (100 µl) Peroxidase-Blocking solution and incubated for 5 minutes. The slides are rinsed in distilled water and placed into a fresh washing buffer bath.

25 Antibody Incubation

Excess of liquid is tapped off and the specimen are covered with 3 drops (100 µl) of Anti-Her-2/neu reagent (Rabbit Anti-Human Her2 Protein in 0.05 mol/L Tris/HCl, 0.1 mol/L NaCl, 15 mmol/L pH7.2 NaN₃ containing stabilizing protein) or negative control reagent (= IGG fraction

of normal rabbit serum at an equivalent protein concentration as the Her2 Ab). After 30 minutes of incubation the slide is rinsed in water and placed into a fresh water bath.

Visualization

5 Excess of liquid is tapped off and the specimen are covered with 3 drops (100 μ l) of visualization reagent. After 30 minutes of incubation the slide is rinsed in water and placed into a fresh water bath. Excess of liquid is tapped off and the specimen are covered with 3 drops (100 μ l) of Substrate-Chromogen solution (DAB) for 10 minutes. After rinsing the specimen with distilled water, photographs are taken with a conventional Olympus microscope to document the staining intensity and tumor regions within the specimen. Optionally a counterstain with hematoxylin was performed.

DNA extraction

15 The whole specimens or dissected subregions are transferred into a microcentrifuge tubes. Optionally a small amount (10 μ l) of preheated TaKaRa solution (DEXPATTM) is preheated and placed onto the specimen to facilitate sample transfer with a scalpel. 50 to 150 μ l of TaKaRa solution were added to the samples depending on the size of the tissue sample selected. The sample are incubated at 100°C for 10 minutes in a block heater, followed by centrifugation at 12.000 rpm in a microcentrifuge. The supernatant is collected using a micropet and placed in a separate microcentrifuge tube. If no deparaffinization step has been undertaken one has to be sure not to withdraw tissue debris and resin. Genomic DNA left in the pellet can be collected by adding 20 resin-free TaKaRa buffer and an additional heating and centrifugation step. Samples are stored at -20°C.

25 Genomic DNA from different tumor cell lines (MCF-7, BT-20, BT-474, SKBR-3, AU-565, UACC-812, UACC-893, HCC-1008, HCC-2157, HCC-1954, HCC-2218, HCC-1937, HCC1599, SW480), or from lymphocytes is prepared with the QIAamp[®] DNA Mini Kits or the QIAamp[®] DNA Blood Mini Kits according to the manufacturers protocol. Usually between 1ng up to 1 μ g DNA is used per reaction.

Quantitative PCR

30 To measure the gene copy number of the genes within the patient samples the respective primer/probes (see table below) are prepared by mixing 25 μ l of the 100 μ M stock solution "Upper Primer", 25 μ l of the 100 μ M stock solution "Lower Primer" with 12,5 μ l of the 100 μ M stock solution Taq Man Probe (Quencher Tamra) and adjusted to 500 μ l with aqua dest. For each

reaction 1,25 µl DNA-Extract of the patient samples or 1,25 µl DNA from the cell lines were mixed with 8,75 µl nuclease-free water and added to one well of a 96 Well-Optical Reaction Plate (Applied Biosystems Part No. 4306737). 1,5 µl Primer/Probe mix, 12, µl Taq Man Universal-PCR Mix (2x) (Applied Biosystems Part No. 4318157) and 1 µl Water are then added. The 96 well plates are closed with 8 Caps/Strips (Applied Biosystems Part Number 4323032) and centrifuged for 3 minutes. Measurements of the PCR reaction are done according to the instructions of the manufacturer with a TaqMan 7900 HT from Applied Biosystems (No. 20114) under appropriate conditions (2 min. 50°C, 10 min. 95°C, 0.15min. 95°C, 1 min. 60°C; 40 cycles). Software SDS 2.0 from Applied Biosystems is used according to the respective instructions. CT-values are then further analyzed with appropriate software (Microsoft Excel™).

EXAMPLE 3

Clinical Samples of patients being treated with Herceptin and a chemotherapeutic agent (e.g. docetaxel, paclitaxel, taxotere, carboplatin, cisplatin, oxaliplatin, vinorelbine) as a second line therapy have been obtained. These samples included formalin-fixed and paraffin-embedded material from primary tumours and metastatic lesions of the respective patients. However, the determination of the ARCHEON genes as disclosed in this invention, has also been performed from fresh tissue after nucleic acid extraction in an independent, neoadjuvant setting. Moreover, whole blood, serum and plasma samples were available for multiple patients.

Multiparametric, clinical assessment of the response to Herceptin in combination with chemotherapeutics (e.g. docetaxel, taxotere, paclitaxel, vinorelbine, carboplatin, cisplatin), or other therapies described below, was performed. Clinical information included histological parameters (TNM-Stage, AJCC grade), standard molecular markers (IHC staining for estrogen receptor, progesteron receptor, Her-2/neu) and sonographical or radiological assessment (e.g. CT). Response to treatment was evaluated according to international standards, i.e. modified WHO criteria and RECIST criteria. Each cancer evaluation in the course of the disease was documented (method and date of evaluation, organ, anatomical description, measurability, size of lesion (longest diameter), greatest perpendicular diameter, tumor area). Moreover, each systemic anticancer therapy including prior chemotherapy with anthracyclins (Doxorubicin or Epirubicin) and/or CMF and the response thereto was evaluated (drug, intent, duration, schedule, number of cycles, cumulative dose). The response to combinatory treatment of metastatic breast cancer patients with Herceptin and chemotherapeutica as second line treatment the modified WHO criteria were used. In addition the initial disease free survival, duration of response and time to progression were taken into consideration. For definition of treatment response standard criteria were used: „Complete Response“ („CR“ = tumor shrinkage of 100 % with no residual disease

being clinical detectable), „Partial Response“ („PR“ = tumor shrinkage of target lesion of at least 50%), „Stable Disease“ („SD“ = tumor shrinkage of less than 50 % or no change) and „Progressive Disease“ („PD“ = tumor growth or new tumor lesions).

More than 70 genes were analyzed according to the method disclosed in example 2 by combining IHC and quantitative PCR or directly by quantitative PCR after nucleic acid extraction from formaldehyde-fixed, paraffin-embedded tissue slides. Results were reconfirmed by independent methodology (VNTR and SNP detection). Alterations of the 43 ARCHEON genes were determined by comparison with reference genes, that are located on the same chromosome (intrachromosomal control,) or different chromosomes (= extrachromosomal control). Intrachromosomal reference genes included MMP28, hKa3 and K20. Extrachromosomal reference genes included GAPDH for chromosome 12. However any other gene not included in the ARCHEONS disclosed in this invention can be used as reference gene for ARCHEON characterization. The reference genes should be independent from the ARCHEON alteration occurring in the neoplastic lesions and should be not affected by chromosomal alterations such as amplifications and deletions. As gene copy numbers of non-amplified genes can be increased in neoplastic lesions due to genomic imbalances such as aneuploidie or polyploidie, each measurement of ARCHEON genes was correlated to multiple reference genes to minimize the influence of genomic imbalances on the relative copy number calculation. Moreover, minimizing systemic errors occurring due to differences in the performance of individual primer/probe pairs were minimized by determining primer/probe performances in control tissues (i.e. non-neoplastic tissues from healthy controls) and euploid control cell lines (e.g. HS68, ATCC #CRL1635). Moreover one well characterized, control cell line was used, that displays aneuploidie for a single chromosome (i.e. Detroit, ATCC#CCL-54; trisomie 21). By measuring genes located on the X-chromosome (e.g. SRY), the Y-chromosome (e.g. Xist) and on chromosome 21, defined copy numbers of 1, 2 and 3 genes could be determined as internal control during each run for standardization. In addition, synthetic targets were spiked into some reactions, that consisted of the target region of the PCR forward and reverse primers of the gene to be normalized, but in between consisted of a synthetic probe hybridization region different from the original probe region of the target gene to be normalized. This allowed internal standardization of each individual qPCR reaction by multiplex PCR. The calculated performance differences were used as a filter for the measurements within the target tissues, i.e. primer/probe differences of each individual gene as depicted in the control cells and tissues were subtracted from each individual gene measurement performed in the target tissue. Thereafter, the individual, filtered CT values were normalized to the different reference genes. Differences between the CT values of the quantitative PCR reactions of the ARCHEON genes and the reference genes remaining after filtering the primer/probe

performance differences were determined and transformed into „copy numbers per cell“. This was done by subtracting the CT values of the target genes from the CT values of the reference genes. The resulting Δ CT values were then transformed in gene copy numbers, with the Δ CT value of the reference gene (Δ CT=0) being defined as „2 copies per cell“, by the following formula:

5 $2^{*(2^{-(\Delta\text{CT}*(-1))})}$. All the calculations were done using standard software (Microsoft Excel™).

References

Patents cited

- | | | |
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Table 1

DNA SEQ ID NO:	Protein SEQ ID NO:	Genbank ID	Unigene_v162_ID	Locus Link ID	Gene Name
1	27	NM_006148.1	Hs.334851	3927	LASP1
2	28	NM_000723.1	Hs.635	782	CACNB1
3	29	NM_000981.1	Hs.381061	6143	RPL19
4	30	Y13467	Hs.15589	5469	PPARGBP
5	31	NM_016507.1	Hs.416108	51755	CrkRS/CRK7
6	32	AB021742.1	Hs.322431	4761	NEUROD2
7	33	NM_006804.1	Hs.77628	10948	MLN64/STARD3
8	34	NM_003673.1	Hs.343603	8557	TELETHONIN
9	35	NM_002686.1	Hs.1892	5409	PNMT
10	36	X03363.1	Hs.446352	2064	ERBB2
11	37	AB008790.1	Hs.86859	2886	GRB7
12	38	NM_002809.1	Hs.9736	5709	PSMD3
13	39	NM_000759.1	Hs.2233	1440	GCSFG/CSF3
14	40	AI023317 NM_014815	Hs.23106	9862	KIAA0130/ TRAP100
15	41	X55005	Hs.724	7067	c-erbA-1 /THRA
16	42	X72631	Hs.2769166	9572	NR1D1
17	43	NM_007359.1	Hs.83422	22794	MLN51
18	44	U77949.1	Hs.405958	990	CDC6
19	45	U41742.1 NM_000964	Hs.361071	5914	RARA
20	46	NM_001067.1	Hs.156346	7153	TOP2A
21	47	NM_001552.1	Hs.1516	3487	IGFBP4
22	48	NM_001838.1	Hs.1652	1236	CCR7 EBI1
23	49	NM_003079.1	Hs.437546	6605	SMARCE1 BAF57
24	50	X14487	Hs.99936	3858	KRT10
25	51	NM_000223.1	Hs.66739	3859	KRT12
26	52	NM_002279.2	Hs.32950	3884	/KRTHA3B
53	76	NM_005937	Hs.497128	4302	MLLT6

Table 1 (continued)

DNA SEQ ID NO:	Protein SEQ ID NO:	Genbank ID	Unigene_v162_ID	Locus Link ID	Gene Name
54	77	XM_008147/ NM_007144	Hs.371617	7703	ZNF144/RNF110
55	78	NM_138687	Hs.9605	8396	PIP5K2B
56	79	NM_020405	Hs.125036	57125	TEM7/PLXDC1
57	80	AF129512	Hs.258579	22806	ZNFN1A3
58	81	XM_085731 NM_133264	Hs.421622	147179	WIRE
59	82	NM_002795	Hs.82793	5691	PSMB3
60	83	NM_033419	Hs.91668	93210	MGC9753 Variant a /CAB2
61	84	NM_033419	Hs.91668	93210	MGC9753 Variant c
62	85	NM_033419	Hs.91668	93210	MGC9753 Variant d
63	86	NM_033419	Hs.91668	93210	MGC9753 Variant e
64	87	NM_033419	Hs.91668	93210	MGC9753 Variant g
65	88	NM_033419	Hs.91668	93210	MGC9753 Variant h
66	89	NM_033419	Hs.91668	93210	MGC9753 Variant i
67	90	AF395708	Hs.133167	94103	ORMDL3
68	91	NM_032875	Hs.194498	84961	MGC15482
69	92	NM_032192	Hs.286192	84152	PPP1R1B
70	93	NM_032339	Hs.333526	84299	MGC14832
71	94	NM_057555 NM_139280	Hs.133167	51242	LOC51242 /ORMDL3
72	95	NM_017748	Hs.406223	54883	FLJ20291
73	96	NM_018530	Hs.306777	55876	Pro2521
74	97	NM_016339	Hs.158530	51195	Link-GEFII

Table 1 (continued)

DNA SEQ ID NO:	Protein SEQ ID NO:	Genbank ID	Unigene_v162_ID	Locus Link ID	Gene Name
75	98	NM_032865	Hs.99037	84951	CTEN
315	393	XM_294897	Hs.270564	30837	NAP4
316	394	NM_032351	Hs.19347	84311	MRLP45
317	395	NM_000458	Hs.408093	6928	TCF2
318	396	NM_152300	Hs.380430	11056	ROK1
319	397	NM_019010	Hs.84905	54474	KRT20
320	398	NM_173213	Hs.9029	25984	KRT23
321	399	NM_033185	Hs.307025	85293	KRTAP3-3
322	400	NM_031959	Hs.307026	83897	KRTAP3-2
323	401	NG_000941		85345	KRTAP3P1
324	402	NM_031958	Hs.307027	83896	KRTAP3-1
325	403	NM_031957	Hs.307030	83895	KRTAP1-5
326	404	NM_030966	Hs.247935	81850	KRTAP1-3
327	405	NM_030967	Hs.247934	81851	KRTAP1-1
328	406	AJ302536		85296	KRTAP2-2
329	407	NM_033184		85294	KRTAP2-4
330	408	NG_000939		85343	KRTAP2P1
331	409	NM_033061	Hs.380164	85287	KRTAP4-7
332	410	NM_033059	Hs.307015	85282	KRTAP4-14
333	411	NM_031854	Hs.307016	83755	KRTAP4-12
334	412	NM_033188	Hs.307016	83755	KRTAP4-5
335	413	NM_033186		85283	KRTAP4-13
336	414	NM_032524	Hs.307022	84616	KRTAP4-4
337	415	NM_033062	Hs.380165	85291	KRTAP4-2
338	416	NM_033060	Hs.380165	85291	KRTAP4-10
339	417	NM_031961	Hs.307013	83899	KRTAP9-2
340	418	NM_031962	Hs.307012	83900	KRTAP9-3
341	419	NM_031963	Hs.307011	83901	KRTAP9-8
342	420	NM_030975	Hs.307010	81870	KRTAP9-9
343	421	NM_033191		85280	KRTAP9-4
344	422	NG_000942		85347	KRTAP9P1
345	423	XM_210345	Hs.463016	85276	KRTAP16-1
346	424	NM_031964	Hs.307009	83902	KRTAP17-1

Table 1 (continued)

DNA SEQ ID NO:	Protein SEQ ID NO:	Genbank ID	Unigene_v162_ID	Locus Link ID	Gene Name
347	425	NM_004138	Hs.197874	3883	KRTHA3A
348	426	NM_002279	Hs.32950	3884	KRTHA3B
349	427	NM_021013	Hs.296942	3885	KRTHA4
350	428	NM_002277	Hs.41696	3881	KRTHA1
351	429	Y16795		8686	KRTHAP1
352	430	NM_003770	Hs.159403	8688	KRTHA7
353	431	NM_006771	Hs.248188	8687	KRTHA8
354	432	NM_002278	Hs.41752	3882	KRTHA2
355	433	NM_002280	Hs.73082	3886	KRTHA5
356	434	NM_003771	Hs.248189	8689	KRTHA6
357	435	NM_002274	Hs.433871	3860	KRT13
358	436	NM_002275	Hs.80342	3866	KRT15
359	437	NM_002276	Hs.309517	3880	KRT19
360	438	NM_000226	Hs.2783	3857	KRT9
361	439	NM_000526	Hs.355214	3861	KRT14
362	440	NM_005557	Hs.432448	3868	KRT16
363	441	NM_000422	Hs.2785	3872	KRT17
364	442	NM_005556	Hs.23881	3855	KRT7
365	443	NG_000944		85349	KRTHBP4
366	444	NG_000943		85348	KRTHBP3
367	445	NM_002281	Hs.170925	3887	KRTHB1
368	446	NM_002284	Hs.278658	3892	KRTHB6
369	447	NM_002282	Hs.182506	3889	KRTHB3
370	448	NG_000940		85344	KRTHBP2
371	449	NM_002283	Hs.182507	3891	KRTHB5
372	450	NM_033045	Hs.272336	3890	KRTHB4
373	451	NM_033033	Hs.134640	3888	KRTHB2
374	452	Y19213		85340	KRTHBP1
375	453	NM_005555	Hs.432677	3854	KRT6B
376	454	NM_173086	Hs.446417	286887	KRT6E
377	455	NM_058242		140446	KRT6C
378	456	NM_005554	Hs.367762	3853	KRT6A
379	457	NM_000424	Hs.433845	3852	KRT5

Table 1 (continued)

DNA SEQ ID NO:	Protein SEQ ID NO:	Genbank ID	Unigene_v162_ID	Locus Link ID	Gene Name
380	458	NM_033448	Hs.55278	112802	KRT6IRS
381	459	NM_175053	Hs.56255	121391	KRT6IRS4
382	460	NM_080747/ AY033495	Hs.147040	140807	K6IRS2/ KRT6
383	461	NM_175068	Hs.319101	55410	KRT6IRS3
384	462	NM_000423	Hs.707	3849	KRT2A
385	463	NM_006121	Hs.80828	3848	KRT1
386	464	NM_057088	Hs.410397	3850	KRT3
387	465	NM_002272	Hs.371139	3851	KRT4
388	466	NM_002273	Hs.356123	3856	KRT8
389	467	NM_000224	Hs.406013	3875	KRT18
390	468	NM_032950	Hs.380710	79148	MMP28
391	469	NM_005419	Hs.72988	6773	STAT2
392	470	NM_002046	Hs.169476	2597	GAPDH

Table 2

DNA SEQ ID NO:	Gene description
1	Member of a subfamily of LIM proteins that contains a LIM domain and an SH3 (Src homology region 3) domain
2	Beta 1 subunit of a voltage-dependent calcium channel (dihydropyridine receptor), involved in coupling of excitation and contraction in muscle, also acts as a calcium channel in various other tissues
3	Ribosomal protein L19, component of the large 60S ribosomal subunit
4	Protein with similarity to nuclear receptor-interacting proteins; binds and co-activates the nuclear receptors PPARalpha (PPARA), RARalpha (RARA), RXR, TRbeta1, and VDR
5	we26e02.x1 CDC2-related protein kinase 7
6	Neurogenic differentiation, a basic-helix-loop-helix transcription factor that mediates neuronal differentiation
7	Protein that is overexpressed in malignant tissues, contains a putative trans-membrane region and a StAR Homology Domain (SHD), may function in steroidogenesis and contribute to tumor progression
8	Telethonin, a sarcomeric protein specifically expressed in skeletal and heart muscle, caps titin (TTN) and is important for structural integrity of the sarcomere
9	Phenylethanolamine N-methyltransferase, acts in catecholamine biosynthesis to convert norepinephrine to epinephrine
10	Tyrosine kinase receptor that has similarity to the EGF receptor, a critical component of IL-6 signaling through the MAP kinase pathway, overexpression associated with prostate, ovary and breast cancer
11	Growth factor receptor-bound protein, an SH2 domain-containing protein that has isoforms which may have a role in cell invasion and metastatic progression of esophageal carcinomas
12	Non-ATPase subunit of the 26S proteasome (prosome, macropain)
13	Granulocyte colony stimulating factor, a glycoprotein that regulates growth, differentiation, and survival of neutrophilic granulocytes

Table 2 (continued)

DNA SEQ ID NO:	Gene description
14	Member of the Vitamin D Receptor Interacting Protein co-activator complex, has strong similarity to thyroid hormone receptor-associated protein (murine Trap100) which function as a transcriptional coregulator
15	Thyroid hormone receptor alpha, a high affinity receptor for thyroid hormone that activates transcription; homologous to avian erythroblastic leukemia virus oncogene
16	encoding Rev-ErbAalp nuclear receptor subfamily 1, group D, member 1
17	Protein that is overexpressed in breast carcinomas
18	Protein which interacts with the DNA replication proteins PCNA and Orc1, translocates from the nucleus following onset of S phase; <i>S. cerevisiae</i> homolog Cdc6p is required for initiation of S phase
19	Retinoic acid receptor alpha, binds retinoic acid and stimulates transcription in a ligand-dependent manner
20	DNA topoisomerase II alpha, member of a family of proteins that relieves torsional stress created by DNA replication, transcription, and cell division;
21	Insulin-like growth factor binding protein, the major IGFBP of osteoblast-like cells, binds IGF1 and IGF2 and inhibits their effects on promoting DNA and glycogen synthesis in osteoblastic cells
22	HUMEBI103 G protein-coupled receptor (EBI 1) gene exon 3 chemokine (C-C motif) receptor 7 G protein-coupled receptor
23	Protein with an HMG 1/2 DNA-binding domain that is subunit of the SNF/SWI complex associated with the nuclear matrix and implicated in regulation of transcription by affecting chromatin structure
24	Keratin 10, a type I keratin that is a component of intermediate filaments and is expressed in terminally differentiated epidermal cells; mutation of the corresponding gene causes epidermolytic hyperkeratosis
25	Keratin 12, a component of intermediate filaments in corneal epithelial cells; mutation of the corresponding gene causes Meesmann corneal dystrophy
26	Hair keratin 3B, a type I keratin that is a member of a family of structural proteins that form intermediate filaments
53	MLLT6 Myeloid/lymphoid or mixed-lineage leukemia (trithorax homolog,

Table 2 (continued)

DNA SEQ ID NO:	Gene description
	Drosophila); translocated to, 6
54	zinc finger protein 144 (Mel-18)
55	Phosphatidylinositol-4-phosphate 5-kinase type II beta isoform a
56	tumor endothelial marker 7 precursor
57	zinc finger protein, subfamily 1A, 3
58	WASP-binding protein putative cr16 and wip like protein similar to Wiskott-Aldrich syndrome protein
59	Proteasome (prosome, macropain) subunit, beta type, 3
60	Predicted
67	ORM1-like 3 (S. cerevisiae)
68	F-box domain A Receptor for Ubiquitination Targets
69	protein phosphatase 1, regulatory (inhibitor) subunit 1B (dopamine and cAMP regulated phosphoprotein, DARPP-32)
70	Predicted Protein
71	Predicted Protein
72	Predicted Protein
73	Predicted Protein
74	Link-GEFII: Link guanine nucleotide exchange factor II
75	C-terminal tensin-like
315	Homo sapiens Nck, Ash and phospholipase C binding protein (NAP4)
316	Homo sapiens mitochondrial ribosomal protein L45 (MRPL45), nuclear gene encoding mitochondrial protein
317	Homo sapiens transcription factor 2, hepatic; LF-B3; variant hepatic nuclear factor (TCF2), transcript variant a
318	Homo sapiens DEAD (Asp-Glu-Ala-Asp) box polypeptide 52 (DDX52)
319	Homo sapiens keratin 20 (KRT20), is a component of intermediate filament network
320	Homo sapiens keratin 23 (histone deacetylase inducible) (KRT23), is a component of intermediate filament network transcript variant 2
321	Homo sapiens keratin associated protein 3-3 (KRTAP3-3) , is a component of intermediate filament network
322	Homo sapiens keratin associated protein 3-2 (KRTAP3-2) ,is a component of

Table 2 (continued)

DNA SEQ ID NO:	Gene description
	intermediate filament network
323	Homo sapiens keratin associated protein 3 pseudogene 1 (KRTAP3P1) on chromosome 17 ,is a component of intermediate filament network
324	Homo sapiens keratin associated protein 3-1 (KRTAP3-1) ,is a component of intermediate filament network
325	Homo sapiens keratin associated protein 1-5 (KRTAP1-5) ,is a component of intermediate filament network
326	Homo sapiens keratin associated protein 1-3 (KRTAP1-3) ,is a component of intermediate filament network
327	Homo sapiens keratin associated protein 1-1 (KRTAP1-1) ,is a component of intermediate filament network
328	HSA302536 Homo sapiens partial mRNA for keratin associated protein KAP2.2 (KRTAP2.2 gene) ,is a component of intermediate filament network
329	Homo sapiens keratin associated protein 2-4 (KRTAP2-4) ,is a component of intermediate filament network
330	Homo sapiens keratin associated protein 2 pseudogene 1 (KRTAP2P1) on chromosome 17, is a component of intermediate filament network
331	Homo sapiens keratin associated protein 4-7 (KRTAP4-7) ,is a component of intermediate filament network
332	Homo sapiens keratin associated protein 4-14 (KRTAP4-14) ,is a component of intermediate filament network
333	Homo sapiens keratin associated protein 4-12 (KRTAP4-12) ,is a component of intermediate filament network
334	Homo sapiens keratin associated protein 4-5 (KRTAP4-5) ,is a component of intermediate filament network
335	Homo sapiens keratin associated protein 4-13 (KRTAP4-13) ,is a component of intermediate filament network
336	Homo sapiens keratin associated protein 4-4 (KRTAP4-4) ,is a component of intermediate filament network
337	Homo sapiens keratin associated protein 4-2 (KRTAP4-2) ,is a component of intermediate filament network
338	Homo sapiens keratin associated protein 4-10 (KRTAP4-10) ,is a component

Table 2 (continued)

DNA SEQ ID NO:	Gene description
	of intermediate filament network
339	Homo sapiens keratin associated protein 9-2 (KRTAP9-2) ,is a component of intermediate filament network
340	Homo sapiens keratin associated protein 9-3 (KRTAP9-3) ,is a component of intermediate filament network
341	Homo sapiens keratin associated protein 9-8 (KRTAP9-8) ,is a component of intermediate filament network
342	Homo sapiens keratin associated protein 9-9 (KRTAP9-9) ,is a component of intermediate filament network
343	Homo sapiens keratin associated protein 9-4 (KRTAP9-4) ,is a component of intermediate filament network
344	Homo sapiens keratin associated protein 9 pseudogene 1 (KRTAP9P1) on chromosome 17 ,is a component of intermediate filament network
345	Homo sapiens keratin associated protein 16-1 (KRTAP16-1) ,is a component of intermediate filament network
346	Homo sapiens keratin associated protein 17-1 (KRTAP17-1) ,is a component of intermediate filament network
347	Homo sapiens keratin, hair, acidic, 3A (KRTHA3A) ,is a component of intermediate filament network
348	Homo sapiens keratin, hair, acidic, 3B (KRTHA3B) ,is a component of intermediate filament network
349	Homo sapiens keratin, hair, acidic, 4 (KRTHA4) ,is a component of intermediate filament network
350	Homo sapiens keratin, hair, acidic, 1 (KRTHA1) ,is a component of intermediate filament network
351	HSA16795 Homo sapiens KRTHAP1 pseudogene,is a component of intermediate filament network
352	Homo sapiens keratin, hair, acidic, 7 (KRTHA7) ,is a component of intermediate filament network
353	Homo sapiens keratin, hair, acidic, 8 (KRTHA8) ,is a component of intermediate filament network
354	Homo sapiens keratin, hair, acidic, 2 (KRTHA2) ,is a component of

Table 2 (continued)

DNA SEQ ID NO:	Gene description
	intermediate filament network
355	Homo sapiens keratin, hair, acidic, 5 (KRTHA5) ,is a component of intermediate filament network
356	Homo sapiens keratin, hair, acidic, 6 (KRTHA6) ,is a component of intermediate filament network
357	Homo sapiens keratin 13 (KRT13), transcript variant 2,is a component of intermediate filament network
358	Homo sapiens keratin 15 (KRT15) ,is a component of intermediate filament network
359	Homo sapiens keratin 19 (KRT19) ,is a component of intermediate filament network
360	Homo sapiens keratin 9 (epidermolytic palmoplantar keratoderma) (KRT9) ,is a component of intermediate filament network
361	Homo sapiens keratin 14 (epidermolysis bullosa simplex, Dowling-Meara, Koebner) (KRT14) ,is a component of intermediate filament network
362	Homo sapiens keratin 16 (focal non-epidermolytic palmoplantar keratoderma) (KRT16) ,is a component of intermediate filament network
363	Homo sapiens keratin 17 (KRT17) ,is a component of intermediate filament network
364	Homo sapiens keratin 7 (KRT7) ,is a component of intermediate filament network
365	Homo sapiens psihHbD hair keratin pseudogene (KRTHBP4) on chromosome 12,is a component of intermediate filament network
366	Homo sapiens psihHbC hair keratin pseudogene (KRTHBP3) on chromosome 12,is a component of intermediate filament network
367	Homo sapiens keratin, hair, basic, 1 (KRTHB1) ,is a component of intermediate filament network
368	Homo sapiens keratin, hair, basic, 6 (monilethrix) (KRTHB6) ,is a component of intermediate filament network
369	Homo sapiens keratin, hair, basic, 3 (KRTHB3) ,is a component of intermediate filament network
370	Homo sapiens psihHbB hair keratin pseudogene (KRTHBP2) on chromosome

Table 2 (continued)

DNA SEQ ID NO:	Gene description
	12 ,is a component of intermediate filament network
371	Homo sapiens keratin, hair, basic, 5 (KRTHB5) ,is a component of intermediate filament network
372	Homo sapiens keratin, hair, basic, 4 (KRTHB4), ,is a component of intermediate filament network
373	Homo sapiens keratin, hair, basic, 2 (KRTHB2) ,is a component of intermediate filament network
374	HSPSIHHBA Homo sapiens putative psihHbA pseudogene for hair keratin, exons 2 to 7
375	Homo sapiens keratin 6B (KRT6B) ,is a component of intermediate filament network
376	Homo sapiens keratin 6E (KRT6E),is a component of intermediate filament network
377	Homo sapiens keratin 6C (KRT6C) ,is a component of intermediate filament network
378	Homo sapiens keratin 6A (KRT6A), ,is a component of intermediate filament network
379	Homo sapiens keratin 5 (epidermolysis bullosa simplex, Dowling-Meara/Kobner/Weber-Cockayne types) (KRT5) ,is a component of intermediate filament network
380	Homo sapiens keratin 6 irs (KRT6IRS) ,is a component of intermediate filament network
381	Homo sapiens keratin 6 irs4 (K6IRS4) ,is a component of intermediate filament network
382	Homo sapiens keratin protein K6irs (K6IRS2) ,is a component of intermediate filament network
383	Homo sapiens keratin protein K6irs (K6IRS2) ,is a component of intermediate filament network
384	Homo sapiens keratin 2A (epidermal ichthyosis bullosa of Siemens) (KRT2A) ,is a component of intermediate filament network
385	Homo sapiens keratin 1 (epidermolytic hyperkeratosis) (KRT1) ,is a component of intermediate filament network

Table 2 (continued)

DNA SEQ ID NO:	Gene description
386	Homo sapiens keratin 3 (KRT3) ,is a component of intermediate filament network
387	Homo sapiens keratin 4 (KRT4) ,is a component of intermediate filament network
388	Homo sapiens keratin 8 (KRT8) ,is a component of intermediate filament network
389	Homo sapiens keratin 18 (KRT18) ,is a component of intermediate filament network
390	Homo sapiens matrix metalloproteinase 28 (MMP28), transcript variant 2
391	Homo sapiens signal transducer and activator of transcription 2, 113kDa (STAT2)
392	Homo sapiens glyceraldehyde-3-phosphate dehydrogenase (GAPD)

Table 3

DNA SEQ ID NO:	Gene function	Subcellular localization
1	SH3/SH2 adapter protein	-
2	voltage-gated calcium channel membrane fraction Channel [passive transporter]	Plasma membrane
3	RNA binding structural protein of ribosome protein biosynthesis	Cytoplasm
4	transcription co-activator nucleus Pol II transcription	Nucleus
5	-	-
6	transcription factor transcription regulation from Pol II promoter neurogenesis	-
7	mitochondrial transport steroid and lipid metabolism	Cytoplasm
8	structural protein of muscle sarcomere alignment	Cytoplasm
9	phenylethanolamine N-methyltransferase Transferase	-
10	New/ErbB-2 receptor receptor signaling protein tyrosine kinase	Plasma membrane
11	SH3/SH2 adapter protein IGF receptor signaling pathway	Cytoplasm
12	26S proteasome Protein degradation Proteasome subunit	Cytoplasm
13	developmental processes positive control of cell proliferation	Extracellular space
14	fatty acid omega-hydroxylase fatty acid omega-hydroxylase	-
15	DNA-binding protein Transcription factor	Nucleus
16	steroid hormone receptor transcription co-repressor	Nucleus
17	-	-
18	nucleotide binding cell cycle regulator DNA replication checkpoint regulation of CDK activity	nucleus

DNA SEQ ID NO:	Gene function	Subcellular localization
19	retinoic acid receptor transcription co-activator transcription factor	nucleus
20	DNA binding DNA topoisomerase (ATP-hydrolyzing)	nucleus
21	skeletal development DNA metabolism signal transduction cell proliferation	
22		plasma membrane
23	chromatin binding transcription co-activator nucleosome disassembly transcription	nucleus nuclear chromosome
24	Cell structure Cytoskeletal Epidermal Development and Maintenance	cytoplasm
25	structural protein vision cell shape and cell size control intermediate filament	cytoplasm
26	cell shape and cell size control Cell structure	cytoplasm
53		-
54	leucine-zipper containing fusion	-
55		
56	Tumor endothelial marker 7 precursor; may be involved in angiogenesis	-
57	Aiolos; DNA binding protein that may be a transcription factor; has strong similarity to murine Znfn1a3, contains zinc finger domain	-
58	The WASP-binding protein WIRE has a role in the regulation of the actin filament system downstream of the platelet-derived growth factor receptor	-
59		-
60		-
61		-

DNA SEQ ID NO:	Gene function	Subcellular localization
67		-
68		-
69	Midbrain dopaminergic neurons play a critical role in multiple brain functions, and abnormal signaling through dopaminergic pathways has been implicated in several major neurologic and psychiatric disorders. One well-studied target for the actions of dopamine is DARPP32.	-
70		-
71		-
72		-
73		-
74	Brain-specific guanine nucleotide exchange factor; activates the ERK/MAP kinase cascade plus R-Ras and H-ras; activates targets through a Ca2+- and diacylglycerol-sensitive mechanism; active protein associates with membranes	-
75	C-terminal tensin-like Phosphotyrosine-binding domain, phosphotyrosine-interaction (PI) domain	-
315		
316		
317		
318		cytoplasm
319	KRT20, integral part of the intermediate filamentous network	Cytoplasm
320	KRT23, integral part of the intermediate filamentous network	Cytoplasm

DNA SEQ ID NO:	Gene function	Subcellular localization
321	KRTAP3-3, integral part of the intermediate filamentous network	Cytoplasm
322	KRTAP3-2, integral part of the intermediate filamentous network	Cytoplasm
323	KRTAP3P1, integral part of the intermediate filamentous network	Cytoplasm
324	KRTAP3-1, integral part of the intermediate filamentous network	Cytoplasm
325	KRTAP1-5, integral part of the intermediate filamentous network	cytoplasm
326	KRTAP1-3, integral part of the intermediate filamentous network	cytoplasm
327	KRTAP1-1, integral part of the intermediate filamentous network	Cytoplasm
328	KRTAP2-2, integral part of the intermediate filamentous network	Cytoplasm
329	KRTAP2-4, integral part of the intermediate filamentous network	Cytoplasm
330	KRTAP2P1, integral part of the intermediate filamentous network	Cytoplasm
331	KRTAP4-7, integral part of the intermediate filamentous network	Cytoplasm
332	KRTAP4-14, integral part of the intermediate filamentous network	Cytoplasm
333	KRTAP4-12, integral part of the intermediate filamentous network	cytoplasm
334	KRTAP4-5, integral part of the intermediate filamentous network	cytoplasm
335	KRTAP4-13, integral part of the intermediate filamentous network	Cytoplasm
336	KRTAP4-4, integral part of the intermediate filamentous network	Cytoplasm
337	KRTAP4-2, integral part of the intermediate filamentous network	Cytoplasm
338	KRTAP4-10, integral part of the intermediate filamentous network	Cytoplasm
339	KRTAP9-2, integral part of the intermediate filamentous network	Cytoplasm
340	KRTAP9-3, integral part of the intermediate filamentous network	Cytoplasm

DNA SEQ ID NO:	Gene function	Subcellular localization
341	KRTAP9-8, integral part of the intermediate filamentous network	cytoplasm
342	KRTAP9-9, integral part of the intermediate filamentous network	cytoplasm
343	KRTAP9-4, integral part of the intermediate filamentous network	Cytoplasm
344	KRTAP9P1, integral part of the intermediate filamentous network	Cytoplasm
345	KRTAP16-1, integral part of the intermediate filamentous network	Cytoplasm
346	KRTAP17-1, integral part of the intermediate filamentous network	Cytoplasm
347	KRTHA3A, integral part of the intermediate filamentous network	Cytoplasm
348	KRTHA3B, integral part of the intermediate filamentous network	Cytoplasm
349	KRTHA4, integral part of the intermediate filamentous network	cytoplasm
350	KRTHA1, integral part of the intermediate filamentous network	cytoplasm
351	KRTHAP1, integral part of the intermediate filamentous network	Cytoplasm
352	KRTHA7, integral part of the intermediate filamentous network	Cytoplasm
353	KRTHA8, integral part of the intermediate filamentous network	Cytoplasm
354	KRTHA2, integral part of the intermediate filamentous network	Cytoplasm
355	KRTHA5, integral part of the intermediate filamentous network	Cytoplasm
356	KRTHA6, integral part of the intermediate filamentous network	Cytoplasm
357	KRT13, integral part of the intermediate filamentous network	cytoplasm
358	KRT15, integral part of the intermediate filamentous network	cytoplasm
359	KRT19, integral part of the intermediate filamentous network	Cytoplasm
360	KRT9, integral part of the intermediate filamentous network	Cytoplasm

DNA SEQ ID NO:	Gene function	Subcellular localization
361	KRT14, integral part of the intermediate filamentous network	Cytoplasm
362	KRT16, integral part of the intermediate filamentous network	Cytoplasm
363	KRT17, integral part of the intermediate filamentous network	Cytoplasm
364	KRT7, integral part of the intermediate filamentous network	Cytoplasm
365	KRTHBP4, integral part of the intermediate filamentous network	cytoplasm
366	KRTHBP3, integral part of the intermediate filamentous network	cytoplasm
367	KRTHB1, integral part of the intermediate filamentous network	Cytoplasm
368	KRTHB6, integral part of the intermediate filamentous network	Cytoplasm
369	KRTHB3, integral part of the intermediate filamentous network	Cytoplasm
370	KRTHBP2, integral part of the intermediate filamentous network	Cytoplasm
371	KRTHB5, integral part of the intermediate filamentous network	Cytoplasm
372	KRTHB4, integral part of the intermediate filamentous network	Cytoplasm
373	KRTHB2, integral part of the intermediate filamentous network	cytoplasm
374	KRTHBP1, integral part of the intermediate filamentous network	cytoplasm
375	KRT6B, integral part of the intermediate filamentous network	Cytoplasm
376	KRT6E, integral part of the intermediate filamentous network	Cytoplasm
377	KRT6C, integral part of the intermediate filamentous network	Cytoplasm
378	KRT6A, integral part of the intermediate filamentous network	Cytoplasm
379	KRT5, integral part of the intermediate filamentous network	Cytoplasm
380	KRT6IRS, integral part of the intermediate filamentous network	Cytoplasm

DNA SEQ ID NO:	Gene function	Subcellular localization
381	KRT6IRS4, integral part of the intermediate filamentous network	cytoplasm
382	KRT6, integral part of the intermediate filamentous network	Cytoplasm
383	KRT6IRS3, integral part of the intermediate filamentous network	Cytoplasm
384	KRT2A, integral part of the intermediate filamentous network	Cytoplasm
385	KRT1, integral part of the intermediate filamentous network	Cytoplasm
386	KRT3, integral part of the intermediate filamentous network	Cytoplasm
387	KRT4, integral part of the intermediate filamentous network	Cytoplasm
388	KRT8, integral part of the intermediate filamentous network	cytoplasm
389	KRT18, integral part of the intermediate filamentous network	Cytoplasm

Table 4

DNA SEQ ID NO:	Protein SEQ ID NO:	Gene Name	DBSNP ID	Type	Codon	AA-Seq
9	34	ERBB2	rs2230698	coding-synon	TCA TCG	S S
9	34	ERBB2	rs2230700	noncoding		
9	34	ERBB2	rs1058808	coding-nonsynon	CCC GCC	P A
9	34	ERBB2	rs1801200	noncoding		
9	34	ERBB2	rs903506	noncoding		
9	34	ERBB2	rs2313170	noncoding		
9	34	ERBB2	rs1136201	coding-nonsynon	ATC GTC	I V
9	34	ERBB2	rs2934968	noncoding		
9	34	ERBB2	rs2172826	noncoding		
9	34	ERBB2	rs1810132	coding-nonsynon	ATC GTC	I V
9	34	ERBB2	rs1801201	noncoding		
14	39	c-erbA-1	rs2230702	coding-synon	TCC TCT	S S
14	39	c-erbA-1	rs2230701	coding-synon	GCC GCT	A A
14	39	c-erbA-1	rs1126503	coding-nonsynon	ACC AGC	T S
14	39	c-erbA-1	rs3471	noncoding		
19	44	TOP2A	rs13695	noncoding		
19	44	TOP2A	rs471692	noncoding		
19	44	TOP2A	rs558068	noncoding		
19	44	TOP2A	rs1064288	noncoding		
19	44	TOP2A	rs1061692	coding-synon	GGA GGG	G G
19	44	TOP2A	rs520630	noncoding		
19	44	TOP2A	rs782774	coding-nonsynon	AAT ATT A TT TTT	N I F
19	44	TOP2A	rs565121	noncoding		
19	44	TOP2A	rs2586112	noncoding		
19	44	TOP2A	rs532299	coding-nonsynon	TTT GTT	F V

Table 4 (continued)

DNA SEQ ID NO:	Protein SEQ ID NO:	Gene Name	DBSNP ID	Type	Codon	AA-Seq
19	44	TOP2A	rs2732786	noncoding		
19	44	TOP2A	rs1804539	noncoding		
19	44	TOP2A	rs1804538	noncoding		
19	44	TOP2A	rs1804537	noncoding		
19	44	TOP2A	rs1141364	coding-synon	AAA AAG	K K
23	48	KRT10	rs12231	noncoding		
23	48	KRT10	rs1132259	coding-nonsynon	CAT CGT	H R
23	48	KRT10	rs1132257	coding-synon	CTG TTG	L L
23	48	KRT10	rs1132256	coding-synon	GCC GCT	A A
23	48	KRT10	rs1132255	coding-synon	CTG TTG	L L
23	48	KRT10	rs1132254	coding-synon	GGC GGT	G G
23	48	KRT10	rs1132252	coding-synon	TTC TTT	F F
23	48	KRT10	rs1132268	coding-nonsynon	CAG GAG	Q E
23	48	KRT10	rs1132258	coding-nonsynon	CGG TGG	R W

Table 5

PRIMER	SEQUENCE		
CACNB1	FAM 5'	CCATATATAAAACCACTGCTCCTTTGTGGCT	3'TAMRA
CACNB1FOR	5'	CCCCCATCTGTCTGTATATTTGTC	3'
CACNB1REV	5'	TGCCTACGCTGACGACTAIGTG	3'
CDC6	FAM 5'	TTTGGTTTCTACAACTGTTGCTAT	3'TAMRA
CDC6 FOR	5'	GGGCTCCACACACCAGATG	3'
CDC6 REV	5'	ACGCTCTGAGCACCCCTCTACA	3'
EBI1-1	FAM 5'	TGTCACAGGACTGAAAACCTCTCCTCATGT	3'TAMRA
EBI1-1 FOR	5'	CCCAAGGCCACGAGCTT	3'
EBI1-1 REV	5'	TGTTGCTCTCTTAAACGAATCGAAA	3'
EBI1-2	FAM 5'	CTGGTCAAAACAACTCTCTGAACCCCTCC	3'TAMRA
EBI1-2 FOR	5'	TGGTGAGGAAAAGCGGACAT	3'
EBI1-2 REV	5'	CTGGCTTGGAGGACAGTGAAG	3'
GCSF	FAM 5'	CAAAGCCCTCCCATCCCATGTAT	3'TAMRA
GCSF FOR	5'	GAGGTGTCGTACCGGTTCTA	3'
GCSF REV	5'	CCGTTCTGCTCTTCCCCTGTCT	3'
GRB7	FAM 5'	CCAGACCCGCTTCACTGACCTGC	3'TAMRA
GRB7 FOR	5'	CGCCTGTACTTCAGCATGGA	3'
GRB7 REV	5'	GCGGTTCAGCTGGTGGAA	3'
HKA3	FAM 5'	ACCCCGAGGCATCACCACAATCAT	3'TAMRA
HKA3 FOR	5'	AGTTCTGCCTCTCTGACAACCAT	3'
HKA3 REV	5'	TAGGCTCAGAGTCAGACCCAAAC	3'
MLN50	FAM 5'	CCCTCGTGGGCTTGTGCTCGG	3'TAMRA
MLN50 FOR	5'	AAGCCGCCAGTTCATCTTTT	3'
MLN50 REV	5'	CTTGTGGTTCAAGTCAAAATGTTTCAG	3'
MLN64-1	FAM 5'	TCTGCCTGGCTCTCGTCGGT	3'TAMRA
MLN64-1 FOR	5'	GGGCTGGGCACCTGACTT	3'

Table 5 (continued)

PRIMER		SEQUENCE	
MLN64-1REV	5'	CCCAACAAGGTCCCAGACT	3'
MLN64-2	FAM 5'	CGGCGCATTGAGCGGCG	3'TAMRA
MLN64-2 FOR	5'	CCCAAGGACTTCGTGAATG	3'
MLN64-2REV	5'	GGCGATCCCTGATGACAAAGTA	3'
PPARBP	FAM 5'	AGCACCAACTGTGAACCAGGTACAATGGC	3'TAMRA
PPARBP FOR	5'	GAGGGAGGCTCTGCTTTGG	3'
PPARBP REV	5'	TCACAACCTAGCGGGTGAGGAG	3'
PSMD3	FAM 5'	TGCAGAGGAACGGCGTGAGCG	3'TAMRA
PSMD3 FOR	5'	TGAGGTTTCTCCCAAAATCGTA	3'
PSMD3 REV	5'	CAGCTCAAGGGAAGCTGTCAATC	3'
RAR	FAM 5'	CCCCACATGTTCCCCAAGATGCT	3'TAMRA
RAR FOR	5'	GGAGGCGCTAAAGGTCTACGT	3'
RAR REV	5'	TGATGCTTCGCAGGTCAGTAA	3'
RPL23A	FAM 5'	CTCCTGCCCTCCTAAAGCTGAAGCC	3'TAMRA
RPL23A FOR	5'	GGACGCGTGGGCTTTTC	3'
RPL23A REV	5'	TGTGGCTGTGGACACCTTTC	3'
RPL19	FAM 5'	CCACAAGCTGAAGGCAGACAAGGCC	3'TAMRA
RPL19 FOR	5'	GCGGATTCTCATGGAACACA	3'
RPL19 REV	5'	GGTCAGCCAGGAGCTTCTTG	3'
NEUROD2	FAM 5'	ACCACCTTGGCAGGTTGTCCAG	3'TAMRA
NEUROD2 FOR	5'	CGCATGCACGACCTGAAC	3'
NEUROD2 REV	5'	GTCTCGATCTTGGACAGCTTCTG	3'
TELE TELETHONIN	FAM 5'	ACACTGTCCACACGCGCCCGAGG	3'TAMRA
TELE TELETHONIN FOR	5'	CTGGGCAGAAATGGAAGGATCT	3'
TELE TELETHONIN REV	5'	GGGACTCTAGCAGACCCACACT	3'
PENT PNMT	FAM 5'	CACCCACCTGGATTCCCTGTTC	3'TAMRA
PENT PNMT FOR	5'	CCTTCAGACAGCGGTAGATGATG	3'
PENT PNMT REV	5'	GGGTATTATTCTTTATTAGTGCCACTT	3'
HER2/NEU;ERBB2	FAM 5'	TTCCCTAAGGCTTTCAGTACCCAGGATCTG	3'TAMRA

Table 5 (continued)

PRIMER	SEQUENCE
HER2/NEU:ERBB FOR	5' CCAGCTGGCCCTTTCCT 3'
HER2/NEU:ERBB REV	5' GAATGGGTGCGCTTTTGTCTTAG 3'
KIA0130	FAM 5' TCACGGACCTCAGCCTGCCCT 3'TAMRA
KIA0130 FOR	5' TGGTGAAGGTGTCAGCCATGT 3'
KIA0130 REV	5' TCAGAGTGCAGCAATGGCTTT 3'
THRA	FAM 5' ACCTCCTTCCCCAGCTCCCC 3'TAMRA
THRA FOR	5' GGCAACATCTTACTTGTCTTTGA 3'
THRA REV	5' CCAAGGAAGCACAGACAATATTC 3'
MLN51	FAM 5' TCCTCCCTATCCATGGCACTAAACCACTTC 3'TAMRA
MLN51 FOR	5' TGGGCAAGGGCTCCTATCT 3'
MLN51 REV	5' GTTACCCCTGGCAGACGTATG 3'
TOP2A	FAM 5' TGCCTCTGAGTCTGAATCTCCCAAAGAGAGA 3'TAMRA
TOP2A FOR	5' GAGTAGTATGTGATTAATTCAGCTCTTGAC 3'
TOP2A REV	5' TCAAATGTTGTCCCGAGTCT 3'
KRT10	FAM 5' CAGAAATTCCGAAGACAGACAAGAACTATTGTCTATGCC 3'TAMRA
	T
KRT10 FOR	5' GATTAGTAACCCATAGCAGTTGAAGGT 3'
KRT10 REV	5' ATTTACTGACGGTGTGCTGAACATAC 3'
K12 KRT12	FAM 5' TGACAGACTCCAAATCACAAGCACAGTCAAC 3'TAMRA
K12 KRT12 FOR	5' TGATGGTTTGGAGGAAAGTTTATTT 3'
K12 KRT12 REV	5' TTTGGTTGGTCTTTAGAGGAATC 3'
NR1D1	FAM 5' TGCCCAACCATGCATCAGGTAGCCC 3'TAMRA
NR1D1 FOR	5' CAGCTCACCTGGCAACTTCA 3'
NR1D1 REV	5' CCTGAATTTCCCAGCGATGT 3'
HSERBT1	FAM 5' CGCCGCTCCCGTTCTGCT 3'TAMRA
HSERBT FOR	5' TGGCCAAGCGTAAGCTGAT 3'
HSERBT REV	5' GCTGCAGTGATCGGATCATCT 3'
MLLT6	FAM 5' CACCATGGAGCCCATCGTGTG 3'TAMRA
MLLT6 FOR	5' ATCCCCGAGGTGCAATTG 3'
MLLT6 REV	5' AGCGATCATGAGGCACGTACT 3'

Table 5 (continued)

PRIMER	SEQUENCE	
ZNF144	FAM 5' CCTGCCAGAGATAGGAGACCCAGACAGCT	3'TAMRA
ZNF144 FOR	5' ATCCCCCTGAGCCTTTTCA	3'
ZNF144 REV	5' CAGCCTCTGGTCCCACCAT	3'
PIP5K2B	FAM 5' TGATCATCAATTCCAAACCTCTCCCCGAA	3'TAMRA
PIP5K2B FOR	5' CCCCATGGTGTCCGAAAC	3'
PIP5K2B REV	5' TGCCAGGAGCCTCCATACC	3'
TEM7	FAM 5' CAGCCTTCTAAAACACAATGTATTTCATGT	3'TAMRA
TEM7 FOR	5' CCTGAACCTTAATGGTAGAATTCAAAAGATC	3'
TEM7 REV	5' TATTAACACTGAGAATCCATGCAGAGA	3'
ZNFN1A3	FAM 5' TATCTGGTCTCAGGGATTGCTCCTATGTATTCAG	3'TAMRA
	C	
ZNFN1A3 FOR	5' CACAGAGCCCTGCTGAAATG	3'
ZNFN1A3 REV	5' GCGAGGTCATTTGGTTTTTAGAAA	3'
WIRE	FAM 5' CTGTGATCCGAAATGGTGCCAG	3'TAMRA
WIRE FOR	5' CCGTCTCCACATCCAAACCT	3'
WIRE REV	5' ACCCATGCATTCGGTATGGT	3'
PSMB3	FAM 5' AGTGGCACCTGCGCCGAACAA	3'TAMRA
PSMB3 FOR	5' CCCCATGGTGACTGATGACTT	3'
PSMB3 REV	5' CCAGAGGGACTCACACATTCC	3'
MGC9753	FAM 5' CCAGAAACTTTCATCCCAAGGCAGTCT	3'TAMRA
MGC9753 FOR	5' CTGCCCCACAGGAATAGAATG	3'
MGC9753 REV	5' AAAAATCCAGTCTGCTTCAACCA	3'
ORMDL3	FAM 5' AGCTGCCCCAGCTCCACGGA	3'TAMRA
ORMDL3 FOR	5' TCCCTGATGAGCGTGCTTATC	3'
ORMDL3 REV	5' TCTCAGTACTTATTGATTCAAAAATCC	3'
MGC15482	FAM 5' TCCAGTGGAAAGCAACCCAGTGTTT	3'TAMRA
MGC15482 FOR	5' CACTTCTAGAGCTACCGTGGAGTCT	3'
MGC15482 REV	5' CCCTCATTGTAAACCCCTTGCT	3'
PPP1R1B	FAM 5' CAGCGTGGCGCAACAACCCA	3'TAMRA
PPP1R1B FOR	5' GGGATTGTTTCGCCACACATA	3'

Table 5 (continued)

PRIMER	SEQUENCE
PPPIR1B REV	5' CCGATGTTAAGGCCCATAGC 3'
MGC14832	FAM 5' TAAATGTCCGGCCAAACATGAGTTCCC 3'TAMRA
MGC14832 FOR	5' CGCAGTGCCCTGGCACAT 3'
MGC14832 REV	5' GACACCCCTGACCTATGGA 3'
LOC51242	FAM 5' CAGTGACCTCTCCCGTTCCCTTGGA 3'TAMRA
LOC51242 FOR	5' TGGTCCCTGTGTCCTCTTC 3'
LOC51242 REV	5' AGGGTCAGGAGGGAGAAAC 3'
FLJ20291	FAM 5' CCAGTGCCACCCGTTAAAGAGTCAA 3'TAMRA
FLJ20291 FOR	5' TTGTGGGACACTCAGTAACTTTGG 3'
FLJ20291 REV	5' ACAAGCACTCCACCCGAGAT 3'
PRO2521	FAM 5' AGTCTGTCTCACTGCCATCGCCA 3'TAMRA
PRO2521 FOR	5' AAGCCTCTGGGTTTTCCTTT 3'
PRO2521 REV	5' CCCACTGGTGACAGGATGGT 3'
Link-GEFII	FAM 5' CATCTGACATCTTCCCGTGGAG 3'TAMRA
Link-GEFII FOR	5' CTTTGCACGATGTCTCAACCA 3'
Link-GEFII REV	5' TTCCCGTGGAGCAGGAA 3'
CTEN	FAM 5' CCGCCGCTAATATGCAACATTAGGG 3'TAMRA
CTEN FOR	5' CGAGTATTCCAAAGCTGGTATCG 3'
CTEN REV	5' ATCACAGAGAGATGGCCCTTATCT 3'
NAP4	FAM 5' TCCGCTCAGTCGCTCTTTCCG 3'TAMRA
NAP4 FOR	5' TCGGAAGGGCTCCTTCAAA 3'
NAP4 REV	5' CACCGTTGCAGCTCTTGGT 3'
MRLP45	FAM 5' CTCCTATCCCTCATGCTATATAAAGAACTACC 3'TAMRA
MRLP45 FOR	5' GGCTGTGGAAGCTTTGAAG 3'
MRLP45 REV	5' TGAGCAGGATGGGAGAGAACA 3'
TCF2	FAM 5' CAAAAGCTGGCCATGGACGCT 3'TAMRA
TCF2 FOR	5' GCAGGAAGGAGGAGGCATC 3'
TCF2 REV	5' CAGGCTGTGAGTCTGGTTGGA 3'
ROK1	FAM 5' CAGCTGGCTTCCATTTCTCTGGCCT 3'TAMRA
ROK1 FOR	5' TGGCAAAACTGGGTTCAGAGA 3'

Table 5 (continued)

PRIMER	SEQUENCE
ROK1 REV	TCGGACCTTGTGGGATG 3'
KRT1	FAM 5' CCGCGCCTAATATGCAACATTAGGG 3'TAMRA
KRT1 FOR	5' CGAGTATTCCAAAGCTGGTATCG 3'
KRT1 REV	5' ATCACAGAGAGATGGCCCTTATCT 3'
KRT5	FAM 5' CCGCGCCTAATATGCAACATTAGGG 3'TAMRA
KRT5 FOR	5' CGAGTATTCCAAAGCTGGTATCG 3'
KRT5 REV	5' ATCACAGAGAGATGGCCCTTATCT 3'
KRT8	FAM 5' CCGCGCCTAATATGCAACATTAGGG 3'TAMRA
KRT8 FOR	5' CGAGTATTCCAAAGCTGGTATCG 3'
KRT8 REV	5' ATCACAGAGAGATGGCCCTTATCT 3'
KRT9	FAM 5' CCGCGCCTAATATGCAACATTAGGG 3'TAMRA
KRT9 FOR	5' CGAGTATTCCAAAGCTGGTATCG 3'
KRT9 REV	5' ATCACAGAGAGATGGCCCTTATCT 3'
KRT10-2	FAM 5' CCGCGCCTAATATGCAACATTAGGG 3'TAMRA
KRT10-2 FOR	5' CGAGTATTCCAAAGCTGGTATCG 3'
KRT10-2 REV	5' ATCACAGAGAGATGGCCCTTATCT 3'
KRT14	FAM 5' CCGCGCCTAATATGCAACATTAGGG 3'TAMRA
KRT14 FOR	5' CGAGTATTCCAAAGCTGGTATCG 3'
KRT14 REV	5' ATCACAGAGAGATGGCCCTTATCT 3'
KRT18	FAM 5' CCGCGCCTAATATGCAACATTAGGG 3'TAMRA
KRT18 FOR	5' CGAGTATTCCAAAGCTGGTATCG 3'
KRT18 REV	5' ATCACAGAGAGATGGCCCTTATCT 3'
KRT19	FAM 5' CCGCGCCTAATATGCAACATTAGGG 3'TAMRA
KRT19 FOR	5' CGAGTATTCCAAAGCTGGTATCG 3'
KRT19 REV	5' ATCACAGAGAGATGGCCCTTATCT 3'
KRT6a/b	FAM 5' CCGCGCCTAATATGCAACATTAGGG 3'TAMRA
KRT6a/b FOR	5' CGAGTATTCCAAAGCTGGTATCG 3'
KRT6a/b REV	5' ATCACAGAGAGATGGCCCTTATCT 3'
KRT20	FAM 5' TGGCGGGAATCCTATTATCAGACTCTGTAATTG 3'TAMRA
	A

Table 5 (continued)

PRIMER		SEQUENCE	
KRT20 FOR	5'	GCAAGAAATCAGCCATAAGAAAGC	3'
KRT20 REV	5'	TTGCAGCTCCTCTGAGTAAACAT	3'

Table 6

No.	ID	forward	reverse	PCR size (bp)	GB ID
1	D17S946	ACAGTCTATCAAGCAGAGAAAATCCT	TGCCGTGCCAGAGAGA	128-142	Z24029
2	D17S1181	GACAACAGAGCGAGACTCCC	GCCCAGCCTGTCACTTATTC	122	-
3	D17S2026	TGGTCATTCGACAACGAA	CAGCATTTGGATGCAATCC	171-318	G05498 X53777
4	D17S838	CTCCAGAAATCCAGACCATGA	AGGACAGTGTGTAGCCCTTC	71-103	Z51080
5	D17S250	GGAAGAAATCAAAATAGACAAT	GCTGGCCATATATATATTTAAACC	151	-
6	D17S1818	CATAGGTATGTTTCAGAAATGTGA	TGCCTACTGGAAACCAGA	119-151	Z52895
7	D17S614	AAGGGGAAGGGCTTTCAAAGCT	NGGAGTTGCAGTGAGCCAAGAT	136	L29873
8	D17S2019	CAAAAGCTTAATGATGCTCAAAACC	TTGTTTCCCTTTGACTTTCTGA	151-152	G07286 Z39013
9	D17S608	TAGGTTCACTCTCATTTTCTTCAG	GTCIGGGTCTTTATGGNGCTTGTG	136	L29870
10	D17S1655	CGGACCAGAGTGTCCATGG	GCATACAGCACCCCTCTACCT	240	-
11	D17S2147	AGGGGAGAAATAAATAAATCTGTGG	CAGGAGTGAGACACTCTCCATG	138	G15195
12	D17S754	TGGATTCACTGACTCAGCCTGC	GCGTGTCTGTCTCCATGTGTGC	145	-
13	D17S1814	TCCCAATGACGGTGATG	CTGGAGGTTGGCTTGTGGAT	150-166	Z52854
14	D17S2007	GGTCCACGAAATTTGCTG	CCACCCAGAAAAACAGGAGA	102-103	G07073 X03438
15	D17S1246	TCGATCTCTGACCTTGTGA	TTGTACCCCAATTCCTTTC	115	-
16	D17S1979	CCTGGATAGATTCAGCTCCC	CTTGTCCTTCTCAATCCTCC	199	G11172 X55068
17	D17S1984	TTAAGCAAGGTTTAAATTAAGCTGC	GATTACAGTGCTCCCTCTCCC	134	G14779 T50487
18	D17S1984	GGTTTAAATTAAGCTGCATGGC	GATTACAGTGCTCCCTCTCCC	126	G11580 T50487
19	D17S1867	AGTTTGACACTGAGGCTTTG	TTTAGACTTGGTAACTGCCG	94	Z51301
20	D17S1788	TGCAGATGCCCTAAGAACTTTTCAG	GCCATGATCTCCCAAAGCC	156-168	Z52160
21	D17S1836	TCGAGGTTATGGTGAGCC	AAACTGTGTGTCAAGGATACT	167-173	Z53182
22	D17S1787	GCTGATCTGAAGCCAAATGA	TACATGAAGGCATGGTCTG	239-251	Z52130
23	D17S1660	CTAATATAATCCTGGGCACATGG	GCTGCGGACCAGACAGAT	201	G06069
24	D17S2154	GATAAAAACAAGCACTGGCTCC	CCCACGGCTTCTTGATCTA	137	G15440
25	D17S1955	TGTAATGTAAGCCCCCATGAGG	CACCTCAACTCAACAGTCTAAAGGTG	180	G11900
26	D17S2098	GTGAGTTCAAGCATAGTAATTATCC	ATTCAGCCTCAGTTCAGTCTTC	181	G13994
27	D17S518	GATCCAGTGGAGACTCAGAG	TAGTCTCTGGGACACCCAGA	88 - 100	X60690
28	D17S1851	ATTCTGAGTGTCTACCTGTGTGAG	ACTGACTGCGCCACTGC	237 - 253	Z53675
29	D11S4358	TCGAGAAGGACAAATCAAC	GAACAGGTTAGTCCATTGG	58	-

Table 6 (continued)

No.	ID	forward	reverse	PCR size (bp)	GB ID
30	D17S964	GTTCCTTCCTCTTGTTGGG	AGTCAGCTGAGATTGTGCC	224	L36695
31	D19S1091	CAAGCCAAAGACATGCCAGTT	CCCCACACACAGCTCATATG	238	G14589
32	D17S1179	TTTCTCTCTCATTCATTGGG	GCAACAGAGGGAGACTCCAA	113 - 125	-
33	D10S2160	TCCCATCCCGTAAGACCTC	TATGGAGTACCTACTCTATGCCAGG	349	G06592
34	D17S1230	ATTCAAAGCTGGATCCCTTT	AGCTGTGACAAATGCCCTGTA	108	L32949
35	D17S1338	TCACCTGAGATTGGGAGACC	AAGATGGGCAGGAATGG	178 - 200	-
36	D17S2011	TCACTGTCCTCCAAAGCCAG	AAACACCACACTCTCCCTTG	115	G07143
37	D17S1237	TTCITGGGCTTCCCGTAGCC	GGGGCAGACGACTTCTCCTT	186	L32947
38	D17S2038	GGGATACAACTTTAAAGTTCC	ATTACCTAATGAGGATTCCTTT	228	G6219
39	D17S2091	GCTGAAATAGCCATCTTGAGCTAC	TCCGCATCCTTTTAAAGAGGCAC	157	G13941
40	D17S649	CTTTCACCTCTTCAGCTGAAGAGG	TGACGTGCTATTTCCTGTTTGTCT	146	L36685
41	D17S1190	GTTTGTGCTATGCCTGC	CAACACACTACCCCAGGA	122	L18197
42	M87506	ACTCCTCAATCIGTAGGGTCT	GAGTCCGCTACCTGAGTGCT	102 - 120	m87506

EPO - Munich
9
28. Okt. 2003CLAIMS

1. A method for the prediction of response to cancer treatment, by the detection of at least markers characterized in that the markers are genes and fragments thereof or genomic nucleic acid sequences that are located on one chromosomal region which is altered in malignant neoplasia.
- 5
2. The method of claim 1 wherein the treatment is an antibody treatment, antihormonal treatment, anti-growth factor treatment, taxol based treatment, anthracycline based treatment and platinum salt based treatment.
3. The method of claim 1 wherein the treatment includes Herceptin™, trastuzumab or 2C2 antibodies.
- 10
4. The method of claim 1 characterized in that the markers are:
- a) genes that are located on one or more chromosomal region(s) which is/are altered in malignant neoplasia; and
- b)
- 15
- i) receptor and ligand; or
- ii) members of the same signal transduction pathway; or
- iii) members of synergistic signal transduction pathways; or
- iv) members of antagonistic signal transduction pathways; or
- v) transcription factor and transcription factor binding site; or
- 20
- vi) integral parts of heteromeric complexes
5. The method of claim 1 or 2 wherein the malignant neoplasia is breast cancer, ovarian cancer, gastric cancer, colon cancer, esophageal cancer, mesenchymal cancer, bladder cancer or non-small cell lung cancer.
6. The method of any of claims 1 to 5 wherein at least one chromosomal region is defined as the cytogenetic region: 1p13, 1q32, 3p21-p24, 5p13-p14, 8q23-q24, 11q13, 12q13, 17q12-q24, 17q11.2-21.3 or 20q13.
- 25
7. A method for the prediction, diagnosis or prognosis of malignant neoplasia by the detection of at least one marker characterized in that the marker is selected from:

- 5
- a) a polynucleotide or polynucleotide analog comprising at least one of the sequences of SEQ ID NO: 319 to 389;
- b) a polynucleotide or polynucleotide analog which hybridizes under stringent conditions to a polynucleotide specified in (a) and encodes a polypeptide exhibiting the same biological function as specified for the respective sequence in Table 2 or 3
- 10
- c) a polynucleotide or polynucleotide analog the sequence of which deviates from the polynucleotide specified in (a) and (c) due to the generation of the genetic code encoding a polypeptide exhibiting the same biological function as specified for the respective sequence in Table 2 or 3
- d) a polynucleotide or polynucleotide analog which represents a specific fragment, derivative or allelic variation of a polynucleotide sequence specified in (a) to (d)
- e) a purified polypeptide encoded by a polynucleotide or polynucleotide analog sequence specified in (a) to (e)
- 15
- f) a purified polypeptide comprising at least one of the sequences of SEQ ID NO: 397 - 467;

Are detected.

8. The method according to any of claims 1 to 6 wherein the markers are selected from:

- 20
- a) a polynucleotide or polynucleotide analog comprising at least one of the sequences of SEQ ID NO: 2 to 6, 8, 9, 11 to 16, 18, 19, 21 to 26, 53 to 76 or 315 to 389
- 25
- b) a polynucleotide or polynucleotide analog which hybridizes under stringent conditions to a polynucleotide specified in (a) and encodes a polypeptide exhibiting the same biological function as specified for the respective sequence in Table 2 or 3
- c) a polynucleotide or polynucleotide analog the sequence of which deviates from the polynucleotide specified in (a) and (b) due to the generation of the genetic code encoding a polypeptide exhibiting the same biological function as specified for the respective sequence in Table 2 or 3

- d) a polynucleotide or polynucleotide analog which represents a specific fragment derivative or allelic variation of a polynucleotide sequence specified in (a) to (c)
 - e) a purified polypeptide encoded by a polynucleotide sequence or polynucleotide analog specified in (a) to (d)
 - 5 f) A purified polypeptide comprising at least one of the sequences of SEQ ID NO: 2 to 52 or 76 to 98 or 393 to 467
- are detected.
9. A diagnostic kit for conducting the method of claims 1 to 8.

**METHODS AND COMPOSITIONS FOR THE PREDICTION, DIAGNOSIS, PROGNOSIS,
PREVENTION AND TREATMENT OF MALIGNANT NEOPLASIA**

ABSTRACT OF THE DISCLOSURE

The invention provides novel compositions, methods and uses, for the prediction., diagnosis, prognosis, prevention and treatment of malignant neoplasia and breast cancer in particular. Genes that are differentially expressed in breast tissue of breast cancer patients versus those of normal people are disclosed.

Figure 1

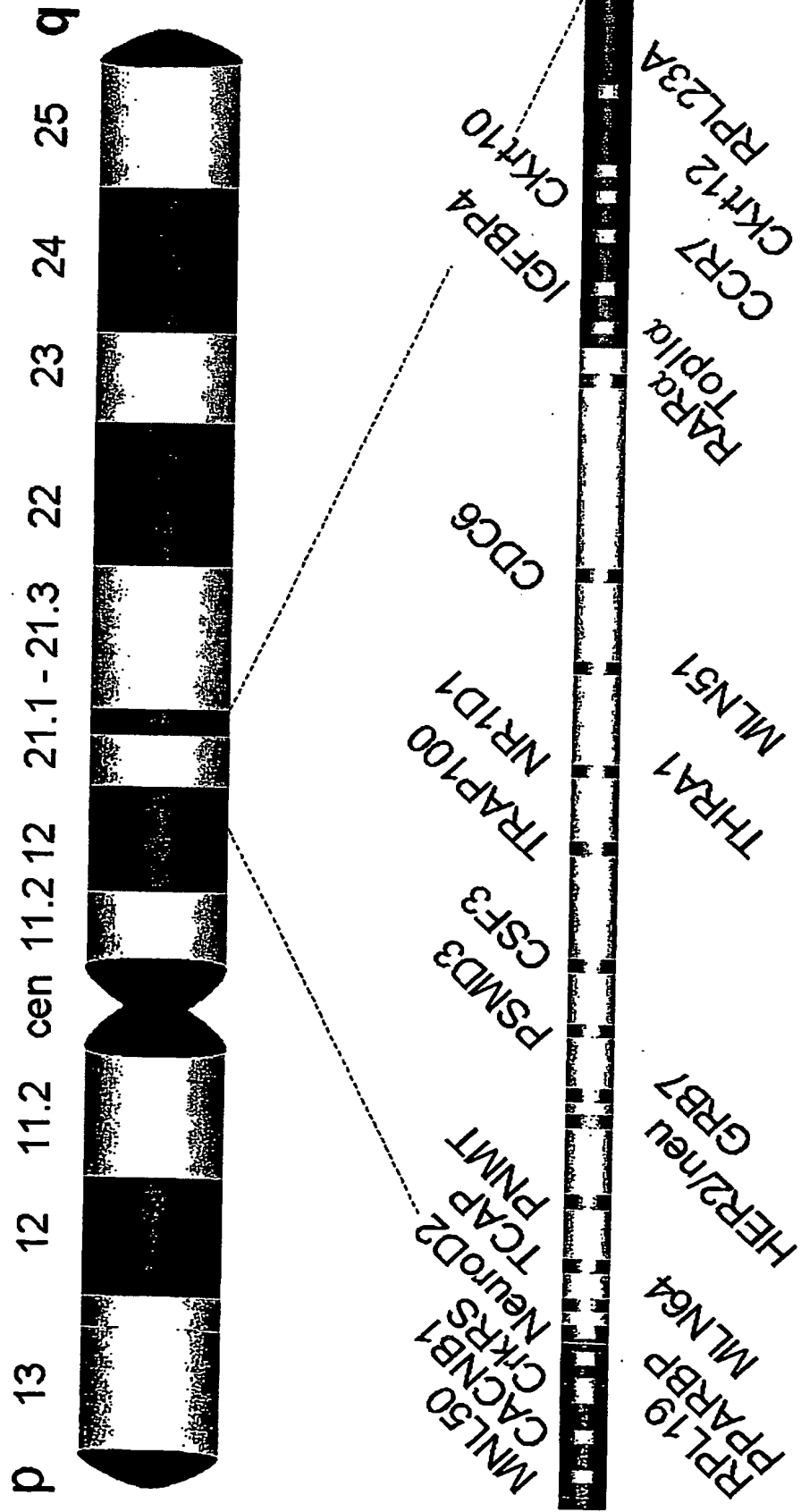
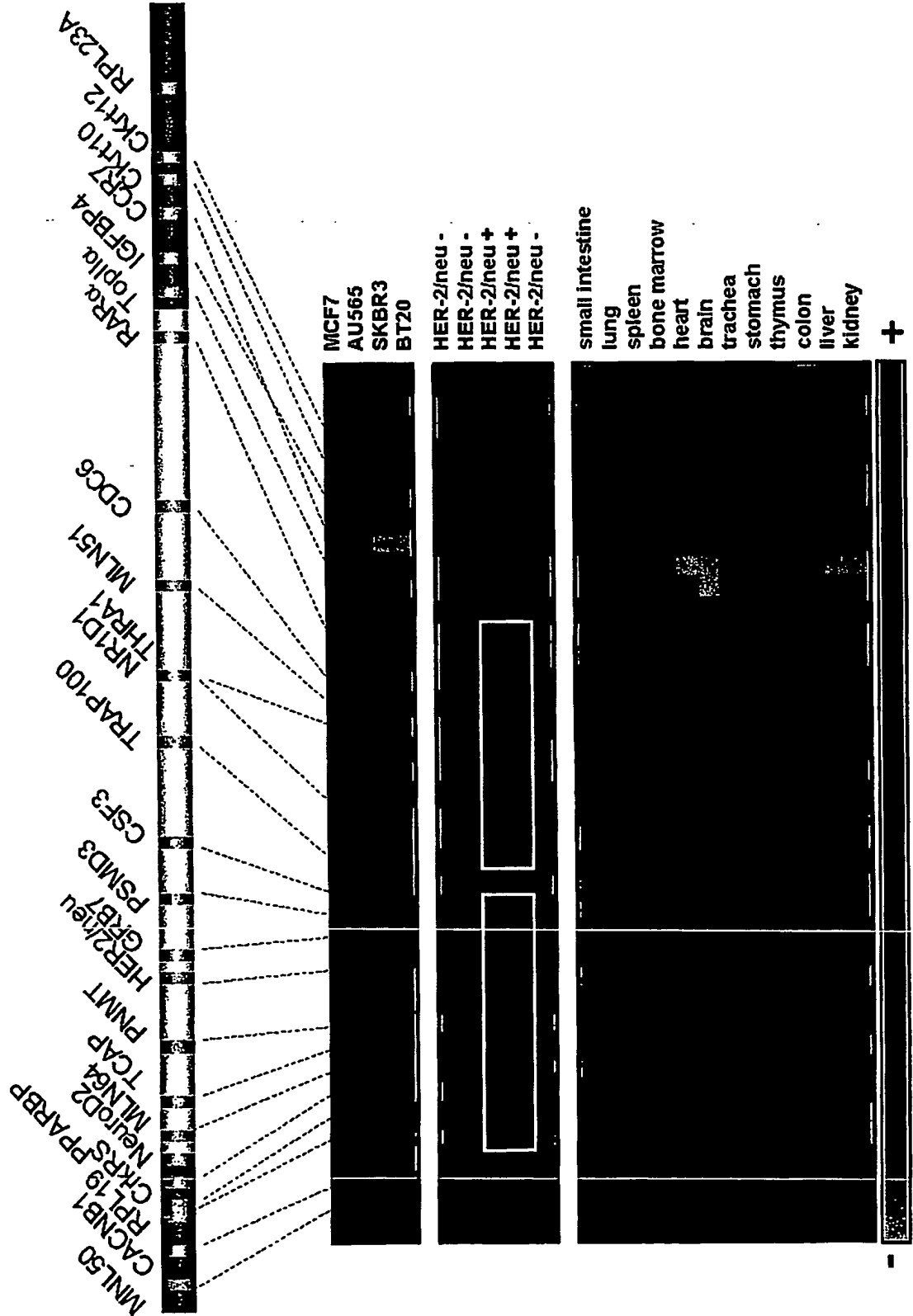


Figure 2



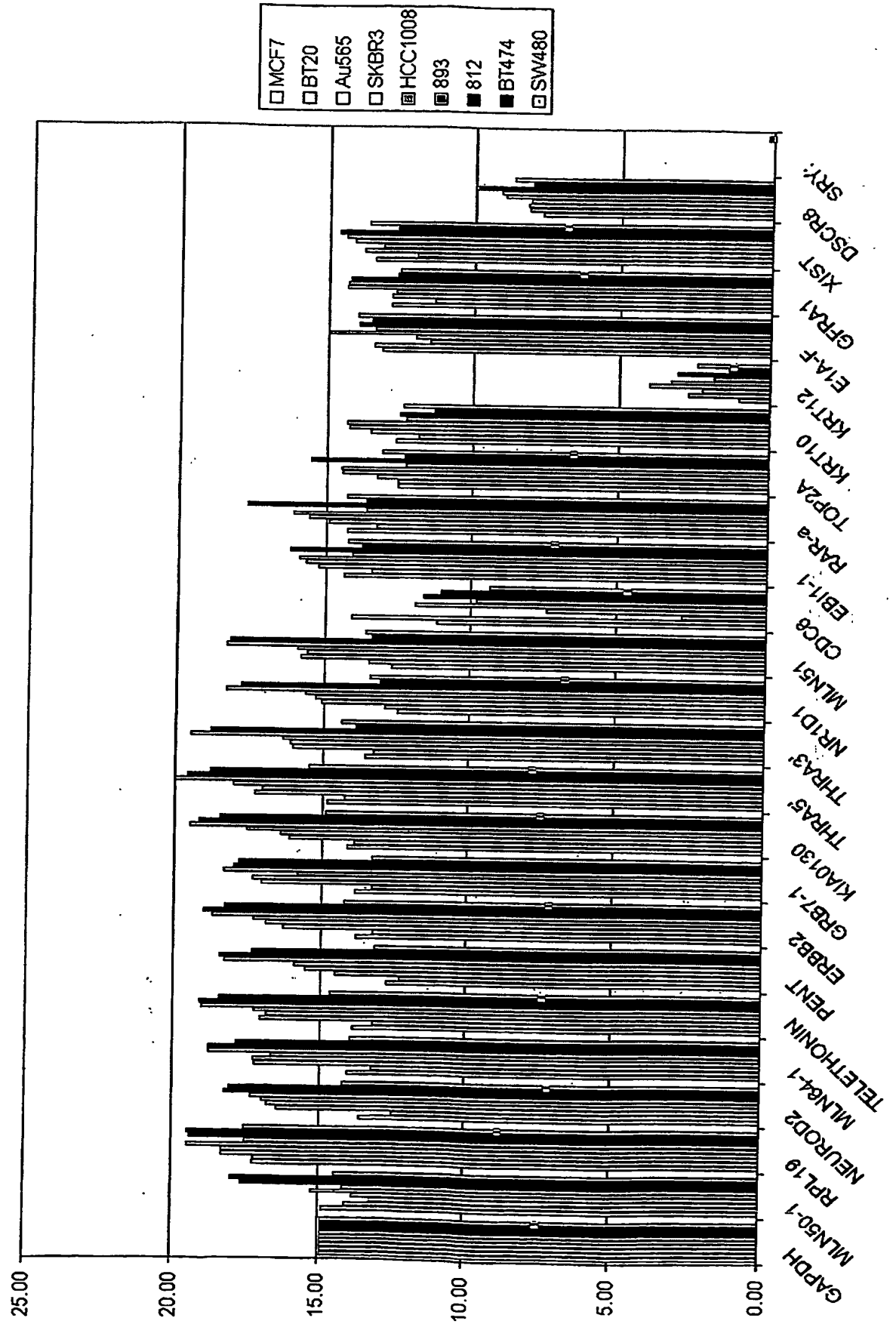


Figure 3

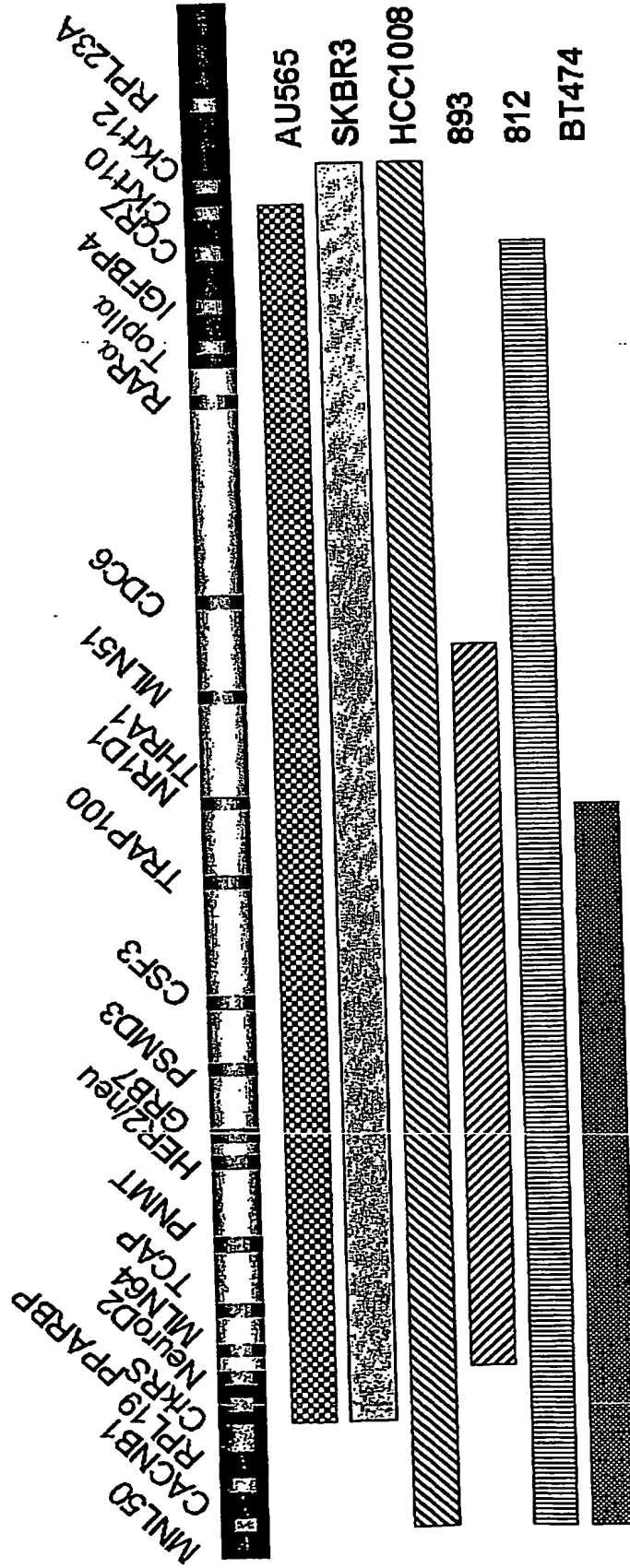


Figure 4

SEQUENCE LISTING

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<120> METHODS AND COMPOSITIONS FOR THE PREDICTION, DIAGNOSIS, PROGNOSIS,
PREVENTION AND TREATMENT OF MALIGNANT NEOPLASIA

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<210> 25

<211> 1871

<212> DNA

<213> Homo sapiens

<400> 25						
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<210> 26

<211> 1447

<212> DNA

<213> Homo sapiens

<400> 26						
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ccagtcctac	ttcaagacca	ttgaggagct	ccagcagaag	atcctgtgca	gcaagtctga	240
gaatgccagg	ctggtggtgc	agatcgacaa	tgccaagctg	gctgcagatg	acttcagaac	300
caagtaccag	acggagcagt	ccctgcggca	gctggtggag	tccgacatca	acagcctgcg	360
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<210> 27

<211> 261

<212> PRT

<213> Homo sapiens

<400> 27

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Lys Val Asn Cys Leu Asp Lys Phe Trp His Lys Ala Cys Phe His Cys
20 25 30
Glu Thr Cys Lys Met Thr Leu Asn Met Lys Asn Tyr Lys Gly Tyr Glu
35 40 45
Lys Lys Pro Tyr Cys Asn Ala His Tyr Pro Lys Gln Ser Phe Thr Met
50 55 60
Val Ala Asp Thr Pro Glu Asn Leu Arg Leu Lys Gln Gln Ser Glu Leu
65 70 75 80
Gln Ser Gln Val Arg Tyr Lys Glu Glu Phe Glu Lys Asn Lys Gly Lys
85 90 95
Gly Phe Ser Val Val Ala Asp Thr Pro Glu Leu Gln Arg Ile Lys Lys
100 105 110
Thr Gln Asp Gln Ile Ser Asn Ile Lys Tyr His Glu Glu Phe Glu Lys
115 120 125
Ser Arg Met Gly Pro Ser Gly Gly Glu Gly Met Glu Pro Glu Arg Arg
130 135 140
Asp Ser Gln Asp Gly Ser Ser Tyr Arg Arg Pro Leu Glu Gln Gln Gln
145 150 155 160
Pro His His Ile Pro Thr Ser Ala Pro Val Tyr Gln Gln Pro Gln Gln
165 170 175
Gln Pro Val Ala Gln Ser Tyr Gly Gly Tyr Lys Glu Pro Ala Ala Pro
180 185 190
Val Ser Ile Gln Arg Ser Ala Pro Gly Gly Gly Lys Arg Tyr Arg
195 200 205
Ala Val Tyr Asp Tyr Ser Ala Ala Asp Glu Asp Glu Val Ser Phe Gln
210 215 220
Asp Gly Asp Thr Ile Val Asn Val Gln Gln Ile Asp Asp Gly Trp Met
225 230 235 240
Tyr Gly Thr Val Glu Arg Thr Gly Asp Thr Gly Met Leu Pro Ala Asn
245 250 255
Tyr Val Glu Ala Ile
260

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<210> 28

<211> 478

<212> PRT

<213> Homo sapiens

<400> 28

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Lys	Arg	Lys	Gly	Arg	Phe	Lys	Arg	Ser	Asp	Gly	Ser	Thr	Ser	Ser	Asp	35	40	45	
Thr	Thr	Ser	Asn	Ser	Phe	Val	Arg	Gln	Gly	Ser	Ala	Glu	Ser	Tyr	Thr	50	55	60	
Ser	Arg	Pro	Ser	Asp	Ser	Asp	Val	Ser	Leu	Glu	Glu	Asp	Arg	Glu	Ala	65	70	75	80
Leu	Arg	Lys	Glu	Ala	Glu	Arg	Gln	Ala	Leu	Ala	Gln	Leu	Glu	Lys	Ala	85	90	95	
Lys	Thr	Lys	Pro	Val	Ala	Phe	Ala	Val	Arg	Thr	Asn	Val	Gly	Tyr	Asn	100	105	110	
Pro	Ser	Pro	Gly	Asp	Glu	Val	Pro	Val	Gln	Gly	Val	Ala	Ile	Thr	Phe	115	120	125	
Glu	Pro	Lys	Asp	Phe	Leu	His	Ile	Lys	Glu	Lys	Tyr	Asn	Asn	Asp	Trp	130	135	140	
Trp	Ile	Gly	Arg	Leu	Val	Lys	Glu	Gly	Cys	Glu	Val	Gly	Phe	Ile	Pro	145	150	155	160
Ser	Pro	Val	Lys	Leu	Asp	Ser	Leu	Arg	Leu	Leu	Gln	Glu	Gln	Lys	Leu	165	170	175	
Arg	Gln	Asn	Arg	Leu	Gly	Ser	Ser	Lys	Ser	Gly	Asp	Asn	Ser	Ser	Ser	180	185	190	
Ser	Leu	Gly	Asp	Val	Val	Thr	Gly	Thr	Arg	Arg	Pro	Thr	Pro	Pro	Ala	195	200	205	
Ser	Ala	Lys	Gln	Lys	Gln	Lys	Ser	Thr	Glu	His	Val	Pro	Pro	Tyr	Asp	210	215	220	
Val	Val	Pro	Ser	Met	Arg	Pro	Ile	Ile	Leu	Val	Gly	Pro	Ser	Leu	Lys	225	230	235	240
Gly	Tyr	Glu	Val	Thr	Asp	Met	Met	Gln	Lys	Ala	Leu	Phe	Asp	Phe	Leu	245	250	255	
Lys	His	Arg	Phe	Asp	Gly	Arg	Ile	Ser	Ile	Thr	Arg	Val	Thr	Ala	Asp	260	265	270	
Ile	Ser	Leu	Ala	Lys	Arg	Ser	Val	Leu	Asn	Asn	Pro	Ser	Lys	His	Ile	275	280	285	
Ile	Ile	Glu	Arg	Ser	Asn	Thr	Arg	Ser	Ser	Leu	Ala	Glu	Val	Gln	Ser	290	295	300	
Glu	Ile	Glu	Arg	Ile	Phe	Glu	Leu	Ala	Arg	Thr	Leu	Gln	Leu	Val	Ala	305	310	315	320
Leu	Asp	Ala	Asp	Thr	Ile	Asn	His	Pro	Ala	Gln	Leu	Ser	Lys	Thr	Ser	325	330	335	
Leu	Ala	Pro	Ile	Ile	Val	Tyr	Ile	Lys	Ile	Thr	Ser	Pro	Lys	Val	Leu	340	345	350	
Gln	Arg	Leu	Ile	Lys	Ser	Arg	Gly	Lys	Ser	Gln	Ser	Lys	His	Leu	Asn	355	360	365	
Val	Gln	Ile	Ala	Ala	Ser	Glu	Lys	Leu	Ala	Gln	Cys	Pro	Pro	Glu	Met	370	375	380	
Phe	Asp	Ile	Ile	Leu	Asp	Glu	Asn	Gln	Leu	Glu	Asp	Ala	Cys	Glu	His	385	390	395	400
Leu	Ala	Glu	Tyr	Leu	Glu	Ala	Tyr	Trp	Lys	Ala	Thr	His	Pro	Pro	Ser	405	410	415	
Ser	Thr	Pro	Pro	Asn	Pro	Leu	Leu	Asn	Arg	Thr	Met	Ala	Thr	Ala	Ala	420	425	430	
Leu	Arg	Arg	Ser	Pro	Ala	Pro	Val	Ser	Asn	Leu	Gln	Val	Gln	Val	Leu	435	440	445	
Thr	Ser	Leu	Arg	Arg	Asn	Leu	Gly	Phe	Trp	Gly	Gly	Leu	Glu	Ser	Ser	450	455	460	

Gln Arg Gly Ser Val Val Pro Gln Glu Gln Glu His Ala Met
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<210> 29

<211> 196

<212> PRT

<213> Homo sapiens

<400> 29

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 Ala Asn Ala Asn Ser Arg Gln Gln Ile Arg Lys Leu Ile Lys Asp Gly
 35 40 45
 Leu Ile Ile Arg Lys Pro Val Thr Val His Ser Arg Ala Arg Cys Arg
 50 55 60
 Lys Asn Thr Leu Ala Arg Arg Lys Gly Arg His Met Gly Ile Gly Lys
 65 70 75 80
 Arg Lys Gly Thr Ala Asn Ala Arg Met Pro Glu Lys Val Thr Trp Met
 85 90 95
 Arg Arg Met Arg Ile Leu Arg Arg Leu Leu Arg Arg Tyr Arg Glu Ser
 100 105 110
 Lys Lys Ile Asp Arg His Met Tyr His Ser Leu Tyr Leu Lys Val Lys
 115 120 125
 Gly Asn Val Phe Lys Asn Lys Arg Ile Leu Met Glu His Ile His Lys
 130 135 140
 Leu Lys Ala Asp Lys Ala Arg Lys Lys Leu Leu Ala Asp Gln Ala Glu
 145 150 155 160
 Ala Arg Arg Ser Lys Thr Lys Glu Ala Arg Lys Arg Arg Glu Glu Arg
 165 170 175
 Leu Gln Ala Lys Lys Glu Glu Ile Ile Lys Thr Leu Ser Lys Glu Glu
 180 185 190
 Glu Thr Lys Lys
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<210> 30

<211> 1566

<212> PRT

<213> Homo sapiens

<400> 30

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 Pro Trp Ser Glu Thr Ile Lys Leu Val Arg Gln Val Met Glu Lys Arg
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 Val Val Met Ser Ser Gly Gly His Gln His Leu Val Ser Cys Leu Glu
 35 40 45
 Thr Leu Gln Lys Ala Leu Lys Val Thr Ser Leu Pro Ala Met Thr Asp
 50 55 60
 Arg Leu Glu Ser Ile Ala Gly Gln Asn Gly Leu Gly Ser His Leu Ser
 65 70 75 80
 Ala Ser Gly Thr Glu Cys Tyr Ile Thr Ser Asp Met Phe Tyr Val Glu
 85 90 95
 Val Gln Leu Asp Pro Ala Gly Gln Leu Cys Asp Val Lys Val Ala His
 100 105 110
 His Gly Glu Asn Pro Val Ser Cys Pro Glu Leu Val Gln Gln Leu Arg
 115 120 125

Glu	Lys	Asn	Ser	Asp	Glu	Phe	Ser	Lys	His	Leu	Lys	Gly	Leu	Val	Asn
130						135					140				
Leu	Tyr	Asn	Leu	Pro	Gly	Asp	Asn	Lys	Leu	Lys	Thr	Lys	Met	Tyr	Leu
145					150					155					160
Ala	Leu	Gln	Ser	Leu	Glu	Gln	Asp	Leu	Ser	Lys	Met	Ala	Ile	Met	Tyr
				165					170						175
Trp	Lys	Ala	Thr	Asn	Ala	Gly	Pro	Leu	Asp	Lys	Ile	Leu	His	Gly	Ser
			180					185					190		
Val	Gly	Tyr	Leu	Thr	Pro	Arg	Ser	Gly	Gly	His	Leu	Met	Asn	Leu	Lys
		195					200					205			
Tyr	Tyr	Val	Ser	Pro	Ser	Asp	Leu	Leu	Asp	Asp	Lys	Thr	Ala	Ser	Pro
	210					215					220				
Ile	Ile	Leu	His	Glu	Asn	Asn	Val	Ser	Arg	Ser	Leu	Gly	Met	Asn	Ala
225					230					235					240
Ser	Val	Thr	Ile	Glu	Gly	Thr	Ser	Ala	Val	Tyr	Lys	Leu	Pro	Ile	Ala
				245					250					255	
Pro	Leu	Ile	Met	Gly	Ser	His	Pro	Val	Asp	Asn	Lys	Trp	Thr	Pro	Ser
			260					265					270		
Phe	Ser	Ser	Ile	Thr	Ser	Ala	Asn	Ser	Val	Asp	Leu	Pro	Ala	Cys	Phe
		275					280					285			
Phe	Leu	Lys	Phe	Pro	Gln	Pro	Ile	Pro	Val	Ser	Arg	Ala	Phe	Val	Gln
	290					295					300				
Lys	Leu	Gln	Asn	Cys	Thr	Gly	Ile	Pro	Leu	Phe	Glu	Thr	Gln	Pro	Thr
305					310					315					320
Tyr	Ala	Pro	Leu	Tyr	Glu	Leu	Ile	Thr	Gln	Phe	Glu	Leu	Ser	Lys	Asp
				325					330					335	
Pro	Asp	Pro	Ile	Pro	Leu	Asn	His	Asn	Met	Arg	Phe	Tyr	Ala	Ala	Leu
			340					345					350		
Pro	Gly	Gln	Gln	His	Cys	Tyr	Phe	Leu	Asn	Lys	Asp	Ala	Pro	Leu	Pro
		355					360					365			
Asp	Gly	Arg	Ser	Leu	Gln	Gly	Thr	Leu	Val	Ser	Lys	Ile	Thr	Phe	Gln
	370					375					380				
His	Pro	Gly	Arg	Val	Pro	Leu	Ile	Leu	Asn	Leu	Ile	Arg	His	Gln	Val
385					390					395					400
Ala	Tyr	Asn	Thr	Leu	Ile	Gly	Ser	Cys	Val	Lys	Arg	Thr	Ile	Leu	Lys
			405						410					415	
Glu	Asp	Ser	Pro	Gly	Leu	Leu	Gln	Phe	Glu	Val	Cys	Pro	Leu	Ser	Glu
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Ser	Arg	Phe	Ser	Val	Ser	Phe	Gln	His	Pro	Val	Asn	Asp	Ser	Leu	Val
		435				440						445			
Cys	Val	Val	Met	Asp	Val	Gln	Gly	Leu	Thr	His	Val	Ser	Cys	Lys	Leu
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Tyr	Lys	Gly	Leu	Ser	Asp	Ala	Leu	Ile	Cys	Thr	Asp	Asp	Phe	Ile	Ala
465					470					475					480
Lys	Val	Val	Gln	Arg	Cys	Met	Ser	Ile	Pro	Val	Thr	Met	Arg	Ala	Ile
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Arg	Arg	Lys	Ala	Glu	Thr	Ile	Gln	Ala	Asp	Thr	Pro	Ala	Leu	Ser	Leu
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Ile	Ala	Glu	Thr	Val	Glu	Asp	Met	Val	Lys	Lys	Asn	Leu	Pro	Pro	Ala
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Ser	Ser	Pro	Gly	Tyr	Gly	Met	Thr	Thr	Gly	Asn	Asn	Pro	Met	Ser	Gly
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Thr	Thr	Thr	Ser	Thr	Asn	Thr	Phe	Pro	Gly	Gly	Pro	Ile	Ala	Thr	Leu
545					550					555					560
Phe	Asn	Met	Ser	Met	Ser	Ile	Lys	Asp	Arg	His	Glu	Ser	Val	Gly	His
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Gly	Glu	Asp	Phe	Ser	Lys	Val	Ser	Gln	Asn	Pro	Ile	Leu	Thr	Ser	Leu
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Pro	Pro	His	His	Thr	Pro	Pro	Pro	Val	Ser	Ser	Met	Ala	Gly	Asn	Thr
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Lys	Asn	His	Pro	Met	Leu	Met	Asn	Leu	Leu	Lys	Asp	Asn	Pro	Ala	Gln
625					630					635					640
Asp	Phe	Ser	Thr	Leu	Tyr	Gly	Ser	Ser	Pro	Leu	Glu	Arg	Gln	Asn	Ser
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Ser	Ser	Gly	Ser	Pro	Arg	Met	Glu	Ile	Cys	Ser	Gly	Ser	Asn	Lys	Thr
			660					665					670		
Lys	Lys	Lys	Lys	Ser	Ser	Arg	Leu	Pro	Pro	Glu	Lys	Pro	Lys	His	Gln
			675				680					685			
Thr	Glu	Asp	Asp	Phe	Gln	Arg	Glu	Leu	Phe	Ser	Met	Asp	Val	Asp	Ser
	690					695					700				
Gln	Asn	Pro	Ile	Phe	Asp	Val	Asn	Met	Thr	Ala	Asp	Thr	Leu	Asp	Thr
705					710					715					720
Pro	His	Ile	Thr	Pro	Ala	Pro	Ser	Gln	Cys	Ser	Thr	Pro	Pro	Thr	Thr
				725					730					735	
Tyr	Pro	Gln	Pro	Val	Pro	His	Pro	Gln	Pro	Ser	Ile	Gln	Arg	Met	Val
		740						745					750		
Arg	Leu	Ser	Ser	Ser	Asp	Ser	Ile	Gly	Pro	Asp	Val	Thr	Asp	Ile	Leu
		755					760					765			
Ser	Asp	Ile	Ala	Glu	Glu	Ala	Ser	Lys	Leu	Pro	Ser	Thr	Ser	Asp	Asp
	770					775					780				
Cys	Pro	Ala	Ile	Gly	Thr	Pro	Leu	Arg	Asp	Ser	Ser	Ser	Ser	Gly	His
785					790					795					800
Ser	Gln	Ser	Thr	Leu	Phe	Asp	Ser	Asp	Val	Phe	Gln	Thr	Asn	Asn	Asn
				805					810					815	
Glu	Asn	Pro	Tyr	Thr	Asp	Pro	Ala	Asp	Leu	Ile	Ala	Asp	Ala	Ala	Gly
			820					825					830		
Ser	Pro	Ser	Ser	Asp	Ser	Pro	Thr	Asn	His	Phe	Phe	His	Asp	Gly	Val
		835					840					845			
Asp	Phe	Asn	Pro	Asp	Leu	Leu	Asn	Ser	Gln	Ser	Gln	Ser	Gly	Phe	Gly
	850					855					860				
Glu	Glu	Tyr	Phe	Asp	Glu	Ser	Ser	Gln	Ser	Gly	Asp	Asn	Asp	Asp	Phe
865					870					875					880
Lys	Gly	Phe	Ala	Ser	Gln	Ala	Leu	Asn	Thr	Leu	Gly	Val	Pro	Met	Leu
			885						890					895	
Gly	Gly	Asp	Asn	Gly	Glu	Thr	Lys	Phe	Lys	Gly	Asn	Asn	Gln	Ala	Asp
			900					905					910		
Thr	Val	Asp	Phe	Ser	Ile	Ile	Ser	Val	Ala	Gly	Lys	Ala	Leu	Ala	Pro
		915						920				925			
Ala	Asp	Leu	Met	Glu	His	His	Ser	Gly	Ser	Gln	Gly	Pro	Leu	Leu	Thr
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Thr	Gly	Asp	Leu	Gly	Lys	Glu	Lys	Thr	Gln	Lys	Arg	Val	Lys	Glu	Gly
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Asn	Gly	Thr	Ser	Asn	Ser	Thr	Leu	Ser	Gly	Pro	Gly	Leu	Asp	Ser	Lys
			965						970					975	
Pro	Gly	Lys	Arg	Ser	Arg	Thr	Pro	Ser	Asn	Asp	Gly	Lys	Ser	Lys	Asp
			980					985				990			
Lys	Pro	Pro	Lys	Arg	Lys	Lys	Ala	Asp	Thr	Glu	Gly	Lys	Ser	Pro	Ser
		995					1000					1005			
His	Ser	Ser	Ser	Asn	Arg	Pro	Phe	Thr	Pro	Pro	Thr	Ser	Thr	Gly	
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Gly	Val	Ala	Thr	Pro	Pro	Ile	Pro	Lys	Ile	Thr	Ile	Gln	Ile	Pro	
	1040					1045					1050				
Lys	Gly	Thr	Val	Met	Val	Gly	Lys	Pro	Ser	Ser	His	Ser	Gln	Tyr	
	1055					1060					1065				
Thr	Ser	Ser	Gly	Ser	Val	Ser	Ser	Ser	Gly	Ser	Lys	Ser	His	His	
	1070					1075					1080				
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	1085					1090					1095				
Met	Lys	Ser	Ser	Lys	Ser	Glu	Gly	Ser	Ser	Ser	Ser	Lys	Leu	Ser	
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Ser	Ser	Met	Tyr	Ser	Ser	Gln	Gly	Ser	Ser	Gly	Ser	Ser	Gln	Ser	
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Lys	Asn	Ser	Ser	Gln	Ser	Gly	Gly	Lys	Pro	Gly	Ser	Ser	Pro	Ile	
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Thr	Lys	His	Gly	Leu	Ser	Ser	Gly	Ser	Ser	Ser	Thr	Lys	Met	Lys	
	1145					1150					1155				
Pro	Gln	Gly	Lys	Pro	Ser	Ser	Leu	Met	Asn	Pro	Ser	Leu	Ser	Lys	
	1160					1165					1170				

Pro	Asn	Ile	Ser	Pro	Ser	His	Ser	Arg	Pro	Pro	Gly	Gly	Ser	Asp
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Lys	Leu	Ala	Ser	Pro	Met	Lys	Pro	Val	Pro	Gly	Thr	Pro	Pro	Ser
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Met	Ser	Gly	Thr	Ser	Ser	Ser	Ser	Gly	Met	Lys	Ser	Ser	Ser	Gly
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Leu	Gly	Ser	Ser	Gly	Ser	Leu	Ser	Gln	Lys	Thr	Pro	Pro	Ser	Ser
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Asn	Ser	Cys	Thr	Ala	Ser	Ser	Ser	Ser	Phe	Ser	Ser	Ser	Gly	Ser
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Ser	Met	Ser	Ser	Ser	Gln	Asn	Gln	His	Gly	Ser	Ser	Lys	Gly	Lys
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Ser	Pro	Ser	Arg	Asn	Lys	Lys	Pro	Ser	Leu	Thr	Ala	Val	Ile	Asp
	1280					1285					1290			
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Lys	Arg	Glu	Lys	Ser	Asp	Lys	Asp	Lys	Ser	Lys	Val	Ser	Thr	Ser
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Gly	Ser	Ser	Val	Asp	Ser	Ser	Lys	Lys	Thr	Ser	Glu	Ser	Lys	Asn
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Val	Gly	Ser	Thr	Gly	Val	Ala	Lys	Ile	Ile	Ile	Ser	Lys	His	Asp
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Gly	Glu	Ser	Ser	Gly	Glu	Gly	Leu	Arg	Pro	Gln	Met	Ala	Ser	Ser
	1400					1405					1410			
Lys	Asn	Tyr	Gly	Ser	Pro	Leu	Ile	Ser	Gly	Ser	Thr	Pro	Lys	His
	1415					1420					1425			
Glu	Arg	Gly	Ser	Pro	Ser	His	Ser	Lys	Ser	Pro	Ala	Tyr	Thr	Pro
	1430					1435					1440			
Gln	Asn	Leu	Asp	Ser	Glu	Ser	Glu	Ser	Gly	Ser	Ser	Ile	Ala	Glu
	1445					1450					1455			
Lys	Ser	Tyr	Gln	Asn	Ser	Pro	Ser	Ser	Asp	Asp	Gly	Ile	Arg	Pro
	1460					1465					1470			
Leu	Pro	Glu	Tyr	Ser	Thr	Glu	Lys	His	Lys	Lys	His	Lys	Lys	Glu
	1475					1480					1485			
Lys	Lys	Lys	Val	Lys	Asp	Lys	Asp	Arg	Asp	Arg	Asp	Arg	Asp	Lys
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Asp	Arg	Asp	Lys	Lys	Lys	Ser	His	Ser	Ile					

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 Ala Ser Gly Thr Leu Gln Pro Ser Ser Gly Gly Gly Ser Ser Asn Ser
 20 25 30
 Arg Glu Arg His Arg Leu Val Ser Lys His Lys Arg His Lys Ser Lys
 35 40 45
 His Ser Lys Asp Met Gly Leu Val Thr Pro Glu Ala Ser Leu Gly
 50 55 60
 Thr Val Ile Lys Pro Leu Val Glu Tyr Asp Asp Ile Ser Ser Asp Ser
 65 70 75 80
 Asp Thr Phe Ser Asp Asp Met Ala Phe Lys Leu Asp Arg Arg Glu Asn
 85 90 95
 Asp Glu Arg Arg Gly Ser Asp Arg Ser Asp Arg Leu His Lys His Arg
 100 105 110
 His His Gln His Arg Arg Ser Arg Asp Leu Leu Lys Ala Lys Gln Thr
 115 120 125
 Glu Lys Glu Lys Ser Gln Glu Val Ser Ser Lys Ser Gly Ser Met Lys
 130 135 140
 Asp Arg Ile Ser Gly Ser Ser Lys Arg Ser Asn Glu Glu Thr Asp Asp
 145 150 155 160
 Tyr Gly Lys Ala Gln Val Ala Lys Ser Ser Ser Lys Glu Ser Arg Ser
 165 170 175
 Ser Lys Leu His Lys Glu Lys Thr Arg Lys Glu Arg Glu Leu Lys Ser
 180 185 190
 Gly His Lys Asp Arg Ser Lys Ser His Arg Lys Arg Glu Thr Pro Lys
 195 200 205
 Ser Tyr Lys Thr Val Asp Ser Pro Lys Arg Arg Ser Arg Ser Pro His
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 Arg Lys Trp Ser Asp Ser Ser Lys Gln Asp Asp Ser Pro Ser Gly Ala
 225 230 235 240
 Ser Tyr Gly Gln Asp Tyr Asp Leu Ser Pro Ser Arg Ser His Thr Ser
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 Ser Asn Tyr Asp Ser Tyr Lys Lys Ser Pro Gly Ser Thr Ser Arg Arg
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 Gln Ser Val Ser Pro Pro Tyr Lys Glu Pro Ser Ala Tyr Gln Ser Ser
 275 280 285
 Thr Arg Ser Pro Ser Pro Tyr Ser Arg Arg Gln Arg Ser Val Ser Pro
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 Tyr Ser Arg Arg Arg Ser Ser Ser Tyr Glu Arg Ser Gly Ser Tyr Ser
 305 310 315 320
 Gly Arg Ser Pro Ser Pro Tyr Gly Arg Arg Arg Ser Ser Ser Pro Phe
 325 330 335
 Leu Ser Lys Arg Ser Leu Ser Arg Ser Pro Leu Pro Ser Arg Lys Ser
 340 345 350
 Met Lys Ser Arg Ser Arg Ser Pro Ala Tyr Ser Arg His Ser Ser Ser
 355 360 365
 His Ser Lys Lys Lys Arg Ser Ser Ser Arg Ser Arg His Ser Ser Ile
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 Ser Pro Val Arg Leu Pro Leu Asn Ser Ser Leu Gly Ala Glu Leu Ser
 385 390 395 400
 Arg Lys Lys Lys Glu Arg Ala Ala Ala Ala Ala Ala Lys Met Asp
 405 410 415
 Gly Lys Glu Ser Lys Gly Ser Pro Val Phe Leu Pro Arg Lys Glu Asn
 420 425 430
 Ser Ser Val Glu Ala Lys Asp Ser Gly Leu Glu Ser Lys Lys Leu Pro
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 Arg Ser Val Lys Leu Glu Lys Ser Ala Pro Asp Thr Glu Leu Val Asn
 450 455 460
 Val Thr His Leu Asn Thr Glu Val Lys Asn Ser Asp Thr Gly Lys
 465 470 475 480
 Val Lys Leu Asp Glu Asn Ser Glu Lys His Leu Val Lys Asp Leu Lys
 485 490 495
 Ala Gln Gly Thr Arg Asp Ser Lys Pro Ile Ala Leu Lys Glu Glu Ile
 500 505 510

Val	Thr	Pro	Lys	Glu	Thr	Glu	Thr	Ser	Glu	Lys	Glu	Thr	Pro	Pro	Pro
		515					520					525			
Leu	Pro	Thr	Ile	Ala	Ser	Pro	Pro	Pro	Pro	Leu	Pro	Thr	Thr	Thr	Pro
		530					535					540			
Pro	Pro	Gln	Thr	Pro	Pro	Leu	Pro	Pro	Leu	Pro	Pro	Ile	Pro	Ala	Leu
		545				550				555					560
Pro	Gln	Gln	Pro	Pro	Leu	Pro	Pro	Ser	Gln	Pro	Ala	Phe	Ser	Gln	Val
				565					570					575	
Pro	Ala	Ser	Ser	Thr	Ser	Thr	Leu	Pro	Pro	Ser	Thr	His	Ser	Lys	Thr
			580					585					590		
Ser	Ala	Val	Ser	Ser	Gln	Ala	Asn	Ser	Gln	Pro	Pro	Val	Gln	Val	Ser
		595					600					605			
Val	Lys	Thr	Gln	Val	Ser	Val	Thr	Ala	Ala	Ile	Pro	His	Leu	Lys	Thr
		610				615					620				
Ser	Thr	Leu	Pro	Pro	Leu	Pro	Leu	Pro	Pro	Leu	Pro	Gly	Gly	Asp	
		625			630					635				640	
Asp	Met	Asp	Ser	Pro	Lys	Glu	Thr	Leu	Pro	Ser	Lys	Pro	Val	Lys	Lys
				645					650					655	
Glu	Lys	Glu	Gln	Arg	Thr	Arg	His	Leu	Leu	Thr	Asp	Leu	Pro	Leu	Pro
			660					665					670		
Pro	Glu	Leu	Pro	Gly	Gly	Asp	Leu	Ser	Pro	Pro	Asp	Ser	Pro	Glu	Pro
		675					680					685			
Lys	Ala	Ile	Thr	Pro	Pro	Gln	Gln	Pro	Tyr	Lys	Lys	Arg	Pro	Lys	Ile
		690				695					700				
Cys	Cys	Pro	Arg	Tyr	Gly	Glu	Arg	Arg	Gln	Thr	Glu	Ser	Asp	Trp	Gly
		705			710					715				720	
Lys	Arg	Cys	Val	Asp	Lys	Phe	Asp	Ile	Ile	Gly	Ile	Ile	Gly	Glu	Gly
			725						730					735	
Thr	Tyr	Gly	Gln	Val	Tyr	Lys	Ala	Arg	Asp	Lys	Asp	Thr	Gly	Glu	Leu
			740					745					750		
Val	Ala	Leu	Lys	Lys	Val	Arg	Leu	Asp	Asn	Glu	Lys	Glu	Gly	Phe	Pro
		755					760					765			
Ile	Thr	Ala	Ile	Arg	Glu	Ile	Lys	Ile	Leu	Arg	Gln	Leu	Ile	His	Arg
		770			775						780				
Ser	Val	Val	Asn	Met	Lys	Glu	Ile	Val	Thr	Asp	Lys	Gln	Asp	Ala	Leu
		785			790					795				800	
Asp	Phe	Lys	Lys	Asp	Lys	Gly	Ala	Phe	Tyr	Leu	Val	Phe	Glu	Tyr	Met
			805						810					815	
Asp	His	Asp	Leu	Met	Gly	Leu	Leu	Glu	Ser	Gly	Leu	Val	His	Phe	Ser
			820					825					830		
Glu	Asp	His	Ile	Lys	Ser	Phe	Met	Lys	Gln	Leu	Met	Glu	Gly	Leu	Glu
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Tyr	Cys	His	Lys	Lys	Asn	Phe	Leu	His	Arg	Asp	Ile	Lys	Cys	Ser	Asn
		850			855						860				
Ile	Leu	Leu	Asn	Asn	Ser	Gly	Gln	Ile	Lys	Leu	Ala	Asp	Phe	Gly	Leu
		865			870					875					880
Ala	Arg	Leu	Tyr	Asn	Ser	Glu	Glu	Ser	Arg	Pro	Tyr	Thr	Asn	Lys	Val
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Ile	Thr	Leu	Trp	Tyr	Arg	Pro	Pro	Glu	Leu	Leu	Leu	Gly	Glu	Glu	Arg
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Tyr	Thr	Pro	Ala	Ile	Asp	Val	Trp	Ser	Cys	Gly	Cys	Ile	Leu	Gly	Glu
		915					920					925			
Leu	Phe	Thr	Lys	Lys	Pro	Ile	Phe	Gln	Ala	Asn	Leu	Glu	Leu	Ala	Gln
		930			935						940				
Leu	Glu	Leu	Ile	Ser	Arg	Leu	Cys	Gly	Ser	Pro	Cys	Pro	Ala	Val	Trp
		945			950					955					960
Pro	Asp	Val	Ile	Lys	Leu	Pro	Tyr	Phe	Asn	Thr	Met	Lys	Pro	Lys	Lys
			965						970					975	
Gln	Tyr	Arg	Arg	Arg	Leu	Arg	Glu	Glu	Phe	Ser	Phe	Ile	Pro	Ser	Ala
			980					985					990		
Ala	Leu	Asp	Leu	Leu	Asp	His	Met	Leu	Thr	Leu	Asp	Pro	Ser	Lys	Arg
		995					1000					1005			
Cys	Thr	Ala	Glu	Gln	Thr	Leu	Gln	Ser	Asp	Phe	Leu	Lys	Asp	Val	
		1010				1015					1020				
Glu	Leu	Ser	Lys	Met	Ala	Pro	Pro	Asp	Leu	Pro	His	Trp	Gln	Asp	
		1025				1030					1035				

Cys	His	Glu	Leu	Trp	Ser	Lys	Lys	Arg	Arg	Arg	Gln	Arg	Gln	Ser
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Gly	Val	Val	Val	Glu	Glu	Pro	Pro	Pro	Ser	Lys	Thr	Ser	Arg	Lys
	1055					1060					1065			
Glu	Thr	Thr	Ser	Gly	Thr	Ser	Thr	Glu	Pro	Val	Lys	Asn	Ser	Ser
	1070					1075					1080			
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	1085					1090					1095			
Gly	Asp	Ala	Ile	Gly	Leu	Ala	Asp	Ile	Thr	Gln	Gln	Leu	Asn	Gln
	1100					1105					1110			
Ser	Glu	Leu	Ala	Val	Leu	Leu	Asn	Leu	Leu	Gln	Ser	Gln	Thr	Asp
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Leu	Ser	Ile	Pro	Gln	Met	Ala	Gln	Leu	Leu	Asn	Ile	His	Ser	Asn
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Pro	Glu	Met	Gln	Gln	Gln	Leu	Glu	Ala	Leu	Asn	Gln	Ser	Ile	Ser
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Ala	Leu	Thr	Glu	Ala	Thr	Ser	Gln	Gln	Gln	Asp	Ser	Glu	Thr	Met
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Ala	Pro	Glu	Glu	Ser	Leu	Lys	Glu	Ala	Pro	Ser	Ala	Pro	Val	Ile
	1175					1180					1185			
Leu	Pro	Ser	Ala	Glu	Gln	Met	Thr	Leu	Glu	Ala	Ser	Ser	Thr	Pro
	1190					1195					1200			
Ala	Asp	Met	Gln	Asn	Ile	Leu	Ala	Val	Leu	Leu	Ser	Gln	Leu	Met
	1205					1210					1215			
Lys	Thr	Gln	Glu	Pro	Ala	Gly	Ser	Leu	Glu	Glu	Asn	Asn	Ser	Asp
	1220					1225					1230			
Lys	Asn	Ser	Gly	Pro	Gln	Gly	Pro	Arg	Arg	Thr	Pro	Thr	Met	Pro
	1235					1240					1245			
Gln	Glu	Glu	Ala	Ala	Ala	Cys	Pro	Pro	His	Ile	Leu	Pro	Pro	Glu
	1250					1255					1260			
Lys	Arg	Pro	Pro	Glu	Pro	Pro	Gly	Pro	Pro	Pro	Pro	Pro	Pro	Pro
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Pro	Pro	Leu	Val	Glu	Gly	Asp	Leu	Ser	Ser	Ala	Pro	Gln	Glu	Leu
	1280					1285					1290			
Asn	Pro	Ala	Val	Thr	Ala	Ala	Leu	Leu	Gln	Leu	Leu	Ser	Gln	Pro
	1295					1300					1305			
Glu	Ala	Glu	Pro	Pro	Gly	His	Leu	Pro	His	Glu	His	Gln	Ala	Leu
	1310					1315					1320			
Arg	Pro	Met	Glu	Tyr	Ser	Thr	Arg	Pro	Arg	Pro	Asn	Arg	Thr	Tyr
	1325					1330					1335			
Gly	Asn	Thr	Asp	Gly	Pro	Glu	Thr	Gly	Phe	Ser	Ala	Ile	Asp	Thr
	1340					1345					1350			
Asp	Glu	Arg	Asn	Ser	Gly	Pro	Ala	Leu	Thr	Glu	Ser	Leu	Val	Gln
	1355					1360					1365			
Thr	Leu	Val	Lys	Asn	Arg	Thr	Phe	Ser	Gly	Ser	Leu	Ser	His	Leu
	1370					1375					1380			
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	1385					1390					1395			
Gly	Asp	Gln	Asp	Leu	Arg	Phe	Ala	Arg	Val	Pro	Leu	Ala	Leu	His
	1400					1405					1410			
Pro	Val	Val	Gly	Gln	Pro	Phe	Leu	Lys	Ala	Glu	Gly	Ser	Ser	Asn
	1415					1420					1425			
Ser	Val	Val	His	Ala	Glu	Thr	Lys	Leu	Gln	Asn	Tyr	Gly	Glu	Leu
	1430					1435					1440			
Gly	Pro	Gly	Thr	Thr	Gly	Ala	Ser	Ser	Ser	Gly	Ala	Gly	Leu	His
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Trp	Gly	Gly	Pro	Thr	Gln	Ser	Ser	Ala	Tyr	Gly	Lys	Leu	Tyr	Arg
	1460					1465					1470			
Gly	Pro	Thr	Arg	Val	Pro	Pro	Arg	Gly	Gly	Arg	Gly	Arg	Gly	Val
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Pro	Tyr													
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<210> 32

<211> 381

<212> PRT

<213> Homo sapiens

<400> 32

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Lys	Phe	Ala	Ser	Trp	Gly	Asp	Gly	Glu	Asp	Asp	Glu	Pro	Arg	Ser	Asp
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Lys	Gly	Asp	Ala	Pro	Pro	Pro	Pro	Pro	Pro	Ala	Pro	Gly	Pro	Gly	Ala
		35					40					45			
Pro	Gly	Pro	Ala	Arg	Ala	Ala	Lys	Pro	Val	Pro	Leu	Arg	Gly	Glu	Glu
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Gly	Thr	Glu	Ala	Thr	Leu	Ala	Glu	Val	Lys	Glu	Glu	Gly	Glu	Leu	Gly
65					70					75					80
Gly	Glu	Glu	Glu	Glu	Glu	Glu	Glu	Glu	Glu	Glu	Gly	Leu	Asp	Glu	Ala
				85					90					95	
Glu	Gly	Glu	Arg	Pro	Lys	Lys	Arg	Gly	Pro	Lys	Lys	Arg	Lys	Met	Thr
			100					105					110		
Lys	Ala	Arg	Leu	Glu	Arg	Ser	Lys	Leu	Arg	Arg	Gln	Lys	Ala	Asn	Ala
			115				120					125			
Arg	Glu	Arg	Asn	Arg	Met	His	Asp	Leu	Asn	Ala	Ala	Leu	Asp	Asn	Leu
	130					135				140					
Arg	Lys	Val	Val	Pro	Cys	Tyr	Ser	Lys	Thr	Gln	Lys	Leu	Ser	Lys	Ile
145					150					155					160
Glu	Thr	Leu	Arg	Leu	Ala	Lys	Asn	Tyr	Ile	Trp	Ala	Leu	Ser	Glu	Ile
				165					170					175	
Leu	Arg	Ser	Gly	Lys	Arg	Pro	Asp	Leu	Val	Ser	Tyr	Val	Gln	Thr	Leu
			180				185						190		
Cys	Lys	Gly	Leu	Ser	Gln	Pro	Thr	Thr	Asn	Leu	Val	Ala	Gly	Cys	Leu
		195					200					205			
Gln	Leu	Asn	Ser	Arg	Asn	Phe	Leu	Thr	Glu	Gln	Gly	Ala	Asp	Gly	Ala
	210					215					220				
Gly	Arg	Phe	His	Gly	Ser	Gly	Gly	Pro	Phe	Ala	Met	His	Pro	Tyr	Pro
225					230					235					240
Tyr	Pro	Cys	Ser	Arg	Leu	Ala	Gly	Ala	Gln	Cys	Gln	Ala	Ala	Gly	Gly
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Leu	Gly	Gly	Gly	Ala	Ala	His	Ala	Leu	Arg	Thr	His	Gly	Tyr	Cys	Ala
			260				265						270		
Ala	Tyr	Glu	Thr	Leu	Tyr	Ala	Ala	Ala	Gly	Gly	Gly	Gly	Ala	Ser	Pro
		275					280					285			
Asp	Tyr	Asn	Ser	Ser	Glu	Tyr	Glu	Gly	Pro	Leu	Ser	Pro	Pro	Leu	Cys
	290					295					300				
Leu	Asn	Gly	Asn	Phe	Ser	Leu	Lys	Gln	Asp	Ser	Ser	Pro	Asp	His	Glu
305					310					315					320
Lys	Ser	Tyr	His	Tyr	Ser	Met	His	Tyr	Ser	Ala	Leu	Pro	Gly	Ser	Arg
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His	Gly	His	Gly	Leu	Val	Phe	Gly	Ser	Ser	Ala	Val	Arg	Gly	Gly	Val
			340				345						350		
His	Ser	Glu	Asn	Leu	Leu	Ser	Tyr	Asp	Met	His	Leu	His	His	Asp	Arg
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Gly	Pro	Met	Tyr	Glu	Glu	Leu	Asn	Ala	Phe	Phe	His	Asn			
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<210> 33

<211> 445

<212> PRT

<213> Homo sapiens

<400> 33

Met	Ser	Lys	Leu	Pro	Arg	Glu	Leu	Thr	Arg	Asp	Leu	Glu	Arg	Ser	Leu
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Pro	Ala	Val	Ala	Ser	Leu	Gly	Ser	Ser	Leu	Ser	His	Ser	Gln	Ser	Leu
			20					25					30		
Ser	Ser	His	Leu	Leu	Pro	Pro	Pro	Glu	Lys	Arg	Arg	Ala	Ile	Ser	Asp
		35					40					45			
Val	Arg	Arg	Thr	Phe	Cys	Leu	Phe	Val	Thr	Phe	Asp	Leu	Leu	Phe	Ile
	50					55					60				
Ser	Leu	Leu	Trp	Ile	Ile	Glu	Leu	Asn	Thr	Asn	Thr	Gly	Ile	Arg	Lys
65					70					75					80
Asn	Leu	Glu	Gln	Glu	Ile	Ile	Gln	Tyr	Asn	Phe	Lys	Thr	Ser	Phe	Phe
				85					90					95	
Asp	Ile	Phe	Val	Leu	Ala	Phe	Phe	Arg	Phe	Ser	Gly	Leu	Leu	Leu	Gly
			100					105					110		
Tyr	Ala	Val	Leu	Gln	Leu	Arg	His	Trp	Trp	Val	Ile	Ala	Val	Thr	Thr
		115					120					125			
Leu	Val	Ser	Ser	Ala	Phe	Leu	Ile	Val	Lys	Val	Ile	Leu	Ser	Glu	Leu
	130					135					140				
Leu	Ser	Lys	Gly	Ala	Phe	Gly	Tyr	Leu	Leu	Pro	Ile	Val	Ser	Phe	Val
145					150					155					160
Leu	Ala	Trp	Leu	Glu	Thr	Trp	Phe	Leu	Asp	Phe	Lys	Val	Leu	Pro	Gln
				165					170					175	
Glu	Ala	Glu	Glu	Glu	Arg	Trp	Tyr	Leu	Ala	Ala	Gln	Val	Ala	Val	Ala
			180					185					190		
Arg	Gly	Pro	Leu	Leu	Phe	Ser	Gly	Ala	Leu	Ser	Glu	Gly	Gln	Phe	Tyr
		195					200					205			
Ser	Pro	Pro	Glu	Ser	Phe	Ala	Gly	Ser	Asp	Asn	Glu	Ser	Asp	Glu	Glu
	210					215					220				
Val	Ala	Gly	Lys	Lys	Ser	Phe	Ser	Ala	Gln	Glu	Arg	Glu	Tyr	Ile	Arg
225					230					235					240
Gln	Gly	Lys	Glu	Ala	Thr	Ala	Val	Val	Asp	Gln	Ile	Leu	Ala	Gln	Glu
				245					250					255	
Glu	Asn	Trp	Lys	Phe	Glu	Lys	Asn	Asn	Glu	Tyr	Gly	Asp	Thr	Val	Tyr
			260					265					270		
Thr	Ile	Glu	Val	Pro	Phe	His	Gly	Lys	Thr	Phe	Ile	Leu	Lys	Thr	Phe
		275					280					285			
Leu	Pro	Cys	Pro	Ala	Glu	Leu	Val	Tyr	Gln	Glu	Val	Ile	Leu	Gln	Pro
	290					295					300				
Glu	Arg	Met	Val	Leu	Trp	Asn	Lys	Thr	Val	Thr	Ala	Cys	Gln	Ile	Leu
305					310					315					320
Gln	Arg	Val	Glu	Asp	Asn	Thr	Leu	Ile	Ser	Tyr	Asp	Val	Ser	Ala	Gly
				325					330					335	
Ala	Ala	Gly	Gly	Val	Val	Ser	Pro	Arg	Asp	Phe	Val	Asn	Val	Arg	Arg
			340					345					350		
Ile	Glu	Arg	Arg	Arg	Asp	Arg	Tyr	Leu	Ser	Ser	Gly	Ile	Ala	Thr	Ser
		355					360					365			
His	Ser	Ala	Lys	Pro	Pro	Thr	His	Lys	Tyr	Val	Arg	Gly	Glu	Asn	Gly
	370					375					380				
Pro	Gly	Gly	Phe	Ile	Val	Leu	Lys	Ser	Ala	Ser	Asn	Pro	Arg	Val	Cys
385					390					395					400
Thr	Phe	Val	Trp	Ile	Leu	Asn	Thr	Asp	Leu	Lys	Gly	Arg	Leu	Pro	Arg
				405					410					415	
Tyr	Leu	Ile	His	Gln	Ser	Leu	Ala	Ala	Thr	Met	Phe	Glu	Phe	Ala	Phe
			420					425					430		
His	Leu	Arg	Gln	Arg	Ile	Ser	Glu	Leu	Gly	Ala	Arg	Ala			
		435					440					445			

<210> 34

<211> 167

<212> PRT

<213> Homo sapiens

<400> 34

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Met Ala Thr Ser Glu Leu Ser Cys Glu Val Ser Glu Glu Asn Cys Glu
1          5          10          15
Arg Arg Glu Ala Phe Trp Ala Glu Trp Lys Asp Leu Thr Leu Ser Thr
          20          25          30
Arg Pro Glu Glu Gly Cys Ser Leu His Glu Glu Asp Thr Gln Arg His
          35          40          45
Glu Thr Tyr His Gln Gln Gly Gln Cys Gln Val Leu Val Gln Arg Ser
          50          55          60
Pro Trp Leu Met Met Arg Met Gly Ile Leu Gly Arg Gly Leu Gln Glu
65          70          75          80
Tyr Gln Leu Pro Tyr Gln Arg Val Leu Pro Leu Pro Ile Phe Thr Pro
          85          90          95
Ala Lys Met Gly Ala Thr Lys Glu Glu Arg Glu Asp Thr Pro Ile Gln
          100          105          110
Leu Gln Glu Leu Leu Ala Leu Glu Thr Ala Leu Gly Gly Gln Cys Val
          115          120          125
Asp Arg Gln Glu Val Ala Glu Ile Thr Lys Gln Leu Pro Pro Val Val
130          135          140
Pro Val Ser Lys Pro Gly Ala Leu Arg Arg Ser Leu Ser Arg Ser Met
145          150          155          160
Ser Gln Glu Ala Gln Arg Gly
          165

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<210> 35

<211> 282

<212> PRT

<213> Homo sapiens

<400> 35

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Met Ser Gly Ala Asp Arg Ser Pro Asn Ala Gly Ala Ala Pro Asp Ser
1          5          10          15
Ala Pro Gly Gln Ala Ala Val Ala Ser Ala Tyr Gln Arg Phe Glu Pro
          20          25          30
Arg Ala Tyr Leu Arg Asn Asn Tyr Ala Pro Pro Arg Gly Asp Leu Cys
          35          40          45
Asn Pro Asn Gly Val Gly Pro Trp Lys Leu Arg Cys Leu Ala Gln Thr
          50          55          60
Phe Ala Thr Gly Glu Val Ser Gly Arg Thr Leu Ile Asp Ile Gly Ser
65          70          75          80
Gly Pro Thr Val Tyr Gln Leu Leu Ser Ala Cys Ser His Phe Glu Asp
          85          90          95
Ile Thr Met Thr Asp Phe Leu Glu Val Asn Arg Gln Glu Leu Gly Arg
          100          105          110
Trp Leu Gln Glu Glu Pro Gly Ala Phe Asn Trp Ser Met Tyr Ser Gln
          115          120          125
His Ala Cys Leu Ile Glu Gly Lys Gly Glu Cys Trp Gln Asp Lys Glu
130          135          140
Arg Gln Leu Arg Ala Arg Val Lys Arg Val Leu Pro Ile Asp Val His
145          150          155          160
Gln Pro Gln Pro Leu Gly Ala Gly Ser Pro Ala Pro Leu Pro Ala Asp
          165          170          175
Ala Leu Val Ser Ala Phe Cys Leu Glu Ala Val Ser Pro Asp Leu Ala
          180          185          190
Ser Phe Gln Arg Ala Leu Asp His Ile Thr Thr Leu Leu Arg Pro Gly
          195          200          205
Gly His Leu Leu Leu Ile Gly Ala Leu Glu Glu Ser Trp Tyr Leu Ala
210          215          220
Gly Glu Ala Arg Leu Thr Val Val Pro Val Ser Glu Glu Glu Val Arg
225          230          235          240

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Glu Ala Leu Val Arg Ser Gly Tyr Lys Val Arg Asp Leu Arg Thr Tyr
 245 250 255
 Ile Met Pro Ala His Leu Gln Thr Gly Val Asp Asp Val Lys Gly Val
 260 265 270
 Phe Phe Ala Trp Ala Gln Lys Val Gly Leu
 275 280

<210> 36

<211> 1255

<212> PRT

<213> Homo sapiens

<400> 36

Met Glu Leu Ala Ala Leu Cys Arg Trp Gly Leu Leu Leu Ala Leu Leu
 1 5 10 15
 Pro Pro Gly Ala Ala Ser Thr Gln Val Cys Thr Gly Thr Asp Met Lys
 20 25 30
 Leu Arg Leu Pro Ala Ser Pro Glu Thr His Leu Asp Met Leu Arg His
 35 40 45
 Leu Tyr Gln Gly Cys Gln Val Val Gln Gly Asn Leu Glu Leu Thr Tyr
 50 55 60
 Leu Pro Thr Asn Ala Ser Leu Ser Phe Leu Gln Asp Ile Gln Glu Val
 65 70 75 80
 Gln Gly Tyr Val Leu Ile Ala His Asn Gln Val Arg Gln Val Pro Leu
 85 90 95
 Gln Arg Leu Arg Ile Val Arg Gly Thr Gln Leu Phe Glu Asp Asn Tyr
 100 105 110
 Ala Leu Ala Val Leu Asp Asn Gly Asp Pro Leu Asn Asn Thr Thr Pro
 115 120 125
 Val Thr Gly Ala Ser Pro Gly Gly Leu Arg Glu Leu Gln Leu Arg Ser
 130 135 140
 Leu Thr Glu Ile Leu Lys Gly Gly Val Leu Ile Gln Arg Asn Pro Gln
 145 150 155 160
 Leu Cys Tyr Gln Asp Thr Ile Leu Trp Lys Asp Ile Phe His Lys Asn
 165 170 175
 Asn Gln Leu Ala Leu Thr Leu Ile Asp Thr Asn Arg Ser Arg Ala Cys
 180 185 190
 His Pro Cys Ser Pro Met Cys Lys Gly Ser Arg Cys Trp Gly Glu Ser
 195 200 205
 Ser Glu Asp Cys Gln Ser Leu Thr Arg Thr Val Cys Ala Gly Gly Cys
 210 215 220
 Ala Arg Cys Lys Gly Pro Leu Pro Thr Asp Cys Cys His Glu Gln Cys
 225 230 235 240
 Ala Ala Gly Cys Thr Gly Pro Lys His Ser Asp Cys Leu Ala Cys Leu
 245 250 255
 His Phe Asn His Ser Gly Ile Cys Glu Leu His Cys Pro Ala Leu Val
 260 265 270
 Thr Tyr Asn Thr Asp Thr Phe Glu Ser Met Pro Asn Pro Glu Gly Arg
 275 280 285
 Tyr Thr Phe Gly Ala Ser Cys Val Thr Ala Cys Pro Tyr Asn Tyr Leu
 290 295 300
 Ser Thr Asp Val Gly Ser Cys Thr Leu Val Cys Pro Leu His Asn Gln
 305 310 315 320
 Glu Val Thr Ala Glu Asp Gly Thr Gln Arg Cys Glu Lys Cys Ser Lys
 325 330 335
 Pro Cys Ala Arg Val Cys Tyr Gly Leu Gly Met Glu His Leu Arg Glu
 340 345 350
 Val Arg Ala Val Thr Ser Ala Asn Ile Gln Glu Phe Ala Gly Cys Lys
 355 360 365
 Lys Ile Phe Gly Ser Leu Ala Phe Leu Pro Glu Ser Phe Asp Gly Asp
 370 375 380

Pro Ala Ser Asn Thr Ala Pro Leu Gln Pro Glu Gln Leu Gln Val Phe
 385 390 395 400
 Glu Thr Leu Glu Glu Ile Thr Gly Tyr Leu Tyr Ile Ser Ala Trp Pro
 405 410 415
 Asp Ser Leu Pro Asp Leu Ser Val Phe Gln Asn Leu Gln Val Ile Arg
 420 425 430
 Gly Arg Ile Leu His Asn Gly Ala Tyr Ser Leu Thr Leu Gln Gly Leu
 435 440 445
 Gly Ile Ser Trp Leu Gly Leu Arg Ser Leu Arg Glu Leu Gly Ser Gly
 450 455 460
 Leu Ala Leu Ile His His Asn Thr His Leu Cys Phe Val His Thr Val
 465 470 475 480
 Pro Trp Asp Gln Leu Phe Arg Asn Pro His Gln Ala Leu Leu His Thr
 485 490 495
 Ala Asn Arg Pro Glu Asp Glu Cys Val Gly Glu Gly Leu Ala Cys His
 500 505 510
 Gln Leu Cys Ala Arg Gly His Cys Trp Gly Pro Gly Pro Thr Gln Cys
 515 520 525
 Val Asn Cys Ser Gln Phe Leu Arg Gly Gln Glu Cys Val Glu Glu Cys
 530 535 540
 Arg Val Leu Gln Gly Leu Pro Arg Glu Tyr Val Asn Ala Arg His Cys
 545 550 555 560
 Leu Pro Cys His Pro Glu Cys Gln Pro Gln Asn Gly Ser Val Thr Cys
 565 570 575
 Phe Gly Pro Glu Ala Asp Gln Cys Val Ala Cys Ala His Tyr Lys Asp
 580 585 590
 Pro Pro Phe Cys Val Ala Arg Cys Pro Ser Gly Val Lys Pro Asp Leu
 595 600 605
 Ser Tyr Met Pro Ile Trp Lys Phe Pro Asp Glu Glu Gly Ala Cys Gln
 610 615 620
 Pro Cys Pro Ile Asn Cys Thr His Ser Cys Val Asp Leu Asp Asp Lys
 625 630 635 640
 Gly Cys Pro Ala Glu Gln Arg Ala Ser Pro Leu Thr Ser Ile Val Ser
 645 650 655
 Ala Val Val Gly Ile Leu Leu Val Val Val Leu Gly Val Val Phe Gly
 660 665 670
 Ile Leu Ile Lys Arg Arg Gln Gln Lys Ile Arg Lys Tyr Thr Met Arg
 675 680 685
 Arg Leu Leu Gln Glu Thr Glu Leu Val Glu Pro Leu Thr Pro Ser Gly
 690 695 700
 Ala Met Pro Asn Gln Ala Gln Met Arg Ile Leu Lys Glu Thr Glu Leu
 705 710 715 720
 Arg Lys Val Lys Val Leu Gly Ser Gly Ala Phe Gly Thr Val Tyr Lys
 725 730 735
 Gly Ile Trp Ile Pro Asp Gly Glu Asn Val Lys Ile Pro Val Ala Ile
 740 745 750
 Lys Val Leu Arg Glu Asn Thr Ser Pro Lys Ala Asn Lys Glu Ile Leu
 755 760 765
 Asp Glu Ala Tyr Val Met Ala Gly Val Gly Ser Pro Tyr Val Ser Arg
 770 775 780
 Leu Leu Gly Ile Cys Leu Thr Ser Thr Val Gln Leu Val Thr Gln Leu
 785 790 795 800
 Met Pro Tyr Gly Cys Leu Leu Asp His Val Arg Glu Asn Arg Gly Arg
 805 810 815
 Leu Gly Ser Gln Asp Leu Leu Asn Trp Cys Met Gln Ile Ala Lys Gly
 820 825 830
 Met Ser Tyr Leu Glu Asp Val Arg Leu Val His Arg Asp Leu Ala Ala
 835 840 845
 Arg Asn Val Leu Val Lys Ser Pro Asn His Val Lys Ile Thr Asp Phe
 850 855 860
 Gly Leu Ala Arg Leu Leu Asp Ile Asp Glu Thr Glu Tyr His Ala Asp
 865 870 875 880
 Gly Gly Lys Val Pro Ile Lys Trp Met Ala Leu Glu Ser Ile Leu Arg
 885 890 895
 Arg Arg Phe Thr His Gln Ser Asp Val Trp Ser Tyr Gly Val Thr Val
 900 905 910

Trp Glu Leu Met Thr Phe Gly Ala Lys Pro Tyr Asp Gly Ile Pro Ala
 915 920 925
 Arg Glu Ile Pro Asp Leu Leu Glu Lys Gly Glu Arg Leu Pro Gln Pro
 930 935 940
 Pro Ile Cys Thr Ile Asp Val Tyr Met Ile Met Val Lys Cys Trp Met
 945 950 955 960
 Ile Asp Ser Glu Cys Arg Pro Arg Phe Arg Glu Leu Val Ser Glu Phe
 965 970 975
 Ser Arg Met Ala Arg Asp Pro Gln Arg Phe Val Val Ile Gln Asn Glu
 980 985 990
 Asp Leu Gly Pro Ala Ser Pro Leu Asp Ser Thr Phe Tyr Arg Ser Leu
 995 1000 1005
 Leu Glu Asp Asp Asp Met Gly Asp Leu Val Asp Ala Glu Glu Tyr
 1010 1015 1020
 Leu Val Pro Gln Gln Gly Phe Phe Cys Pro Asp Pro Ala Pro Gly
 1025 1030 1035
 Ala Gly Gly Met Val His His Arg His Arg Ser Ser Ser Thr Arg
 1040 1045 1050
 Ser Gly Gly Gly Asp Leu Thr Leu Gly Leu Glu Pro Ser Glu Glu
 1055 1060 1065
 Glu Ala Pro Arg Ser Pro Leu Ala Pro Ser Glu Gly Ala Gly Ser
 1070 1075 1080
 Asp Val Phe Asp Gly Asp Leu Gly Met Gly Ala Ala Lys Gly Leu
 1085 1090 1095
 Gln Ser Leu Pro Thr His Asp Pro Ser Pro Leu Gln Arg Tyr Ser
 1100 1105 1110
 Glu Asp Pro Thr Val Pro Leu Pro Ser Glu Thr Asp Gly Tyr Val
 1115 1120 1125
 Ala Pro Leu Thr Cys Ser Pro Gln Pro Glu Tyr Val Asn Gln Pro
 1130 1135 1140
 Asp Val Arg Pro Gln Pro Pro Ser Pro Arg Glu Gly Pro Leu Pro
 1145 1150 1155
 Ala Ala Arg Pro Ala Gly Ala Thr Leu Glu Arg Ala Lys Thr Leu
 1160 1165 1170
 Ser Pro Gly Lys Asn Gly Val Val Lys Asp Val Phe Ala Phe Gly
 1175 1180 1185
 Gly Ala Val Glu Asn Pro Glu Tyr Leu Thr Pro Gln Gly Gly Ala
 1190 1195 1200
 Ala Pro Gln Pro His Pro Pro Pro Ala Phe Ser Pro Ala Phe Asp
 1205 1210 1215
 Asn Leu Tyr Tyr Trp Asp Gln Asp Pro Pro Glu Arg Gly Ala Pro
 1220 1225 1230
 Pro Ser Thr Phe Lys Gly Thr Pro Thr Ala Glu Asn Pro Glu Tyr
 1235 1240 1245
 Leu Gly Leu Asp Val Pro Val
 1250 1255

<210> 37

<211> 532

<212> PRT

<213> Homo sapiens

<400> 37

Met Glu Leu Asp Leu Ser Pro Pro His Leu Ser Ser Ser Pro Glu Asp
 1 5 10 15
 Leu Trp Pro Ala Pro Gly Thr Pro Pro Gly Thr Pro Arg Pro Pro Asp
 20 25 30
 Thr Pro Leu Pro Glu Glu Val Lys Arg Ser Gln Pro Leu Leu Ile Pro
 35 40 45
 Thr Thr Gly Arg Lys Leu Arg Glu Glu Glu Arg Arg Ala Thr Ser Leu
 50 55 60

Pro Ser Ile Pro Asn Pro Phe Pro Glu Leu Cys Ser Pro Pro Ser Gln
 65 70 75 80
 Ser Pro Ile Leu Gly Gly Pro Ser Ser Ala Arg Gly Leu Leu Pro Arg
 85 90 95
 Asp Ala Ser Arg Pro His Val Val Lys Val Tyr Ser Glu Asp Gly Ala
 100 105 110
 Cys Arg Ser Val Glu Val Ala Ala Gly Ala Thr Ala Arg His Val Cys
 115 120 125
 Glu Met Leu Val Gln Arg Ala His Ala Leu Ser Asp Glu Thr Trp Gly
 130 135 140
 Leu Val Glu Cys His Pro His Leu Ala Leu Glu Arg Gly Leu Glu Asp
 145 150 155 160
 His Glu Ser Val Val Glu Val Gln Ala Ala Trp Pro Val Gly Gly Asp
 165 170 175
 Ser Arg Phe Val Phe Arg Lys Asn Phe Ala Lys Tyr Glu Leu Phe Lys
 180 185 190
 Ser Ser Pro His Ser Leu Phe Pro Glu Lys Met Val Ser Ser Cys Leu
 195 200 205
 Asp Ala His Thr Gly Ile Ser His Glu Asp Leu Ile Gln Asn Phe Leu
 210 215 220
 Asn Ala Gly Ser Phe Pro Glu Ile Gln Gly Phe Leu Gln Leu Arg Gly
 225 230 235 240
 Ser Gly Arg Lys Leu Trp Lys Arg Phe Phe Cys Phe Leu Arg Arg Ser
 245 250 255
 Gly Leu Tyr Tyr Ser Thr Lys Gly Thr Ser Lys Asp Pro Arg His Leu
 260 265 270
 Gln Tyr Val Ala Asp Val Asn Glu Ser Asn Val Tyr Val Val Thr Gln
 275 280 285
 Gly Arg Lys Leu Tyr Gly Met Pro Thr Asp Phe Gly Phe Cys Val Lys
 290 295 300
 Pro Asn Lys Leu Arg Asn Gly His Lys Gly Leu Arg Ile Phe Cys Ser
 305 310 315 320
 Glu Asp Glu Gln Ser Arg Thr Cys Trp Leu Ala Ala Phe Arg Leu Phe
 325 330 335
 Lys Tyr Gly Val Gln Leu Tyr Lys Asn Tyr Gln Gln Ala Gln Ser Arg
 340 345 350
 His Leu His Pro Ser Cys Leu Gly Ser Pro Pro Leu Arg Ser Ala Ser
 355 360 365
 Asp Asn Thr Leu Val Ala Met Asp Phe Ser Gly His Ala Gly Arg Val
 370 375 380
 Ile Glu Asn Pro Arg Glu Ala Leu Ser Val Ala Leu Glu Glu Ala Gln
 385 390 395 400
 Ala Trp Arg Lys Lys Thr Asn His Arg Leu Ser Leu Pro Met Pro Ala
 405 410 415
 Ser Gly Thr Ser Leu Ser Ala Ala Ile His Arg Thr Gln Leu Trp Phe
 420 425 430
 His Gly Arg Ile Ser Arg Glu Glu Ser Gln Arg Leu Ile Gly Gln Gln
 435 440 445
 Gly Leu Val Asp Gly Leu Phe Leu Val Arg Glu Ser Gln Arg Asn Pro
 450 455 460
 Gln Gly Phe Val Leu Ser Leu Cys His Leu Gln Lys Val Lys His Tyr
 465 470 475 480
 Leu Ile Leu Pro Ser Glu Glu Glu Gly Arg Leu Tyr Phe Ser Met Asp
 485 490 495
 Asp Gly Gln Thr Arg Phe Thr Asp Leu Leu Gln Leu Val Glu Phe His
 500 505 510
 Gln Leu Asn Arg Gly Ile Leu Pro Cys Leu Leu Arg His Cys Cys Thr
 515 520 525
 Arg Val Ala Leu
 530
 <210> 38
 <211> 534
 <212> PRT

<213> Homo sapiens

<400> 38

Met	Lys	Gln	Glu	Gly	Ser	Ala	Arg	Arg	Arg	Gly	Ala	Asp	Lys	Ala	Lys
1				5					10					15	
Pro	Pro	Pro	Gly	Gly	Gly	Glu	Gln	Glu	Pro	Pro	Pro	Pro	Pro	Ala	Pro
			20					25					30		
Gln	Asp	Val	Glu	Met	Lys	Glu	Glu	Ala	Ala	Thr	Gly	Gly	Gly	Ser	Thr
		35					40					45			
Gly	Glu	Ala	Asp	Gly	Lys	Thr	Ala	Ala	Ala	Ala	Val	Glu	His	Ser	Gln
	50					55					60				
Arg	Glu	Leu	Asp	Thr	Val	Thr	Leu	Glu	Asp	Ile	Lys	Glu	His	Val	Lys
65					70					75					80
Gln	Leu	Glu	Lys	Ala	Val	Ser	Gly	Lys	Glu	Pro	Arg	Phe	Val	Leu	Arg
			85						90					95	
Ala	Leu	Arg	Met	Leu	Pro	Ser	Thr	Ser	Arg	Arg	Leu	Asn	His	Tyr	Val
			100					105					110		
Leu	Tyr	Lys	Ala	Val	Gln	Gly	Phe	Thr	Ser	Asn	Asn	Ala	Thr	Arg	
		115					120					125			
Asp	Phe	Leu	Leu	Pro	Phe	Leu	Glu	Glu	Pro	Met	Asp	Thr	Glu	Ala	Asp
	130					135					140				
Leu	Gln	Phe	Arg	Pro	Arg	Thr	Gly	Lys	Ala	Ala	Ser	Thr	Pro	Leu	Leu
145					150					155					160
Pro	Glu	Val	Glu	Ala	Tyr	Leu	Gln	Leu	Leu	Val	Val	Ile	Phe	Met	Met
			165						170					175	
Asn	Ser	Lys	Arg	Tyr	Lys	Glu	Ala	Gln	Lys	Ile	Ser	Asp	Asp	Leu	Met
		180						185					190		
Gln	Lys	Ile	Ser	Thr	Gln	Asn	Arg	Arg	Ala	Leu	Asp	Leu	Val	Ala	Ala
	195					200						205			
Lys	Cys	Tyr	Tyr	Tyr	His	Ala	Arg	Val	Tyr	Glu	Phe	Leu	Asp	Lys	Leu
	210					215					220				
Asp	Val	Val	Arg	Ser	Phe	Leu	His	Ala	Arg	Leu	Arg	Thr	Ala	Thr	Leu
225					230					235					240
Arg	His	Asp	Ala	Asp	Gly	Gln	Ala	Thr	Leu	Leu	Asn	Leu	Leu	Leu	Arg
			245						250					255	
Asn	Tyr	Leu	His	Tyr	Ser	Leu	Tyr	Asp	Gln	Ala	Glu	Lys	Leu	Val	Ser
		260						265					270		
Lys	Ser	Val	Phe	Pro	Glu	Gln	Ala	Asn	Asn	Asn	Glu	Trp	Ala	Arg	Tyr
		275					280					285			
Leu	Tyr	Tyr	Thr	Gly	Arg	Ile	Lys	Ala	Ile	Gln	Leu	Glu	Tyr	Ser	Glu
	290					295					300				
Ala	Arg	Arg	Thr	Met	Thr	Asn	Ala	Leu	Arg	Lys	Ala	Pro	Gln	His	Thr
305					310					315					320
Ala	Val	Gly	Phe	Lys	Gln	Thr	Val	His	Lys	Leu	Leu	Ile	Val	Val	Glu
			325						330					335	
Leu	Leu	Leu	Gly	Glu	Ile	Pro	Asp	Arg	Leu	Gln	Phe	Arg	Gln	Pro	Ser
			340					345					350		
Leu	Lys	Arg	Ser	Leu	Met	Pro	Tyr	Phe	Leu	Leu	Thr	Gln	Ala	Val	Arg
	355						360					365			
Thr	Gly	Asn	Leu	Ala	Lys	Phe	Asn	Gln	Val	Leu	Asp	Gln	Phe	Gly	Glu
	370					375					380				
Lys	Phe	Gln	Ala	Asp	Gly	Thr	Tyr	Thr	Leu	Ile	Ile	Arg	Leu	Arg	His
385					390					395					400
Asn	Val	Ile	Lys	Thr	Gly	Val	Arg	Met	Ile	Ser	Leu	Ser	Tyr	Ser	Arg
			405						410				415		
Ile	Ser	Leu	Ala	Asp	Ile	Ala	Gln	Lys	Leu	Gln	Leu	Asp	Ser	Pro	Glu
		420						425				430			
Asp	Ala	Glu	Phe	Ile	Val	Ala	Lys	Gly	Ile	Arg	Asp	Gly	Val	Ile	Glu
	435						440					445			
Ala	Ser	Ile	Asn	His	Glu	Lys	Gly	Tyr	Val	Gln	Ser	Lys	Glu	Met	Ile
	450					455					460				
Asp	Ile	Tyr	Ser	Thr	Arg	Glu	Pro	Gln	Leu	Ala	Phe	His	Gln	Arg	Ile
465					470					475					480

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Ser Phe Cys Leu Asp Ile His Asn Met Ser Val Lys Ala Met Arg Phe
 485 490 495
 Pro Pro Lys Ser Tyr Asn Lys Asp Leu Glu Ser Ala Glu Glu Arg Arg
 500 505 510
 Glu Arg Glu Gln Gln Asp Leu Glu Phe Ala Lys Glu Met Ala Glu Asp
 515 520 525
 Asp Asp Asp Ser Phe Pro
 530
 <210> 39
 <211> 207
 <212> PRT
 <213> Homo sapiens

<400> 39
 Met Ala Gly Pro Ala Thr Gln Ser Pro Met Lys Leu Met Ala Leu Gln
 1 5 10 15
 Leu Leu Leu Trp His Ser Ala Leu Trp Thr Val Gln Glu Ala Thr Pro
 20 25 30
 Leu Gly Pro Ala Ser Ser Leu Pro Gln Ser Phe Leu Leu Lys Cys Leu
 35 40 45
 Glu Gln Val Arg Lys Ile Gln Gly Asp Gly Ala Ala Leu Gln Glu Lys
 50 55 60
 Leu Val Ser Glu Cys Ala Thr Tyr Lys Leu Cys His Pro Glu Glu Leu
 65 70 75 80
 Val Leu Leu Gly His Ser Leu Gly Ile Pro Trp Ala Pro Leu Ser Ser
 85 90 95
 Cys Pro Ser Gln Ala Leu Gln Leu Ala Gly Cys Leu Ser Gln Leu His
 100 105 110
 Ser Gly Leu Phe Leu Tyr Gln Gly Leu Leu Gln Ala Leu Glu Gly Ile
 115 120 125
 Ser Pro Glu Leu Gly Pro Thr Leu Asp Thr Leu Gln Leu Asp Val Ala
 130 135 140
 Asp Phe Ala Thr Thr Ile Trp Gln Gln Met Glu Glu Leu Gly Met Ala
 145 150 155 160
 Pro Ala Leu Gln Pro Thr Gln Gly Ala Met Pro Ala Phe Ala Ser Ala
 165 170 175
 Phe Gln Arg Arg Ala Gly Gly Val Leu Val Ala Ser His Leu Gln Ser
 180 185 190
 Phe Leu Glu Val Ser Tyr Arg Val Leu Arg His Leu Ala Gln Pro
 195 200 205
 <210> 40
 <211> 989
 <212> PRT
 <213> Homo sapiens

<400> 40
 Met Lys Val Val Asn Leu Lys Gln Ala Ile Leu Gln Ala Trp Lys Glu
 1 5 10 15
 Arg Trp Ser Tyr Tyr Gln Trp Ala Ile Asn Met Lys Lys Phe Phe Pro
 20 25 30
 Lys Gly Ala Thr Trp Asp Ile Leu Asn Leu Ala Asp Ala Leu Leu Glu
 35 40 45
 Gln Ala Met Ile Gly Pro Ser Pro Asn Pro Leu Ile Leu Ser Tyr Leu
 50 55 60
 Lys Tyr Ala Ile Ser Ser Gln Met Val Ser Tyr Ser Ser Val Leu Thr
 65 70 75 80

Ala	Ile	Ser	Lys	Phe	Asp	Asp	Phe	Ser	Arg	Asp	Leu	Cys	Val	Gln	Ala
				85					90					95	
Leu	Leu	Asp	Ile	Met	Asp	Met	Phe	Cys	Asp	Arg	Leu	Ser	Cys	His	Gly
			100					105					110		
Lys	Ala	Glu	Glu	Cys	Ile	Gly	Leu	Cys	Arg	Ala	Leu	Leu	Ser	Ala	Leu
		115					120					125			
His	Trp	Leu	Leu	Arg	Cys	Thr	Ala	Ala	Ser	Ala	Glu	Arg	Leu	Arg	Glu
	130					135					140				
Gly	Leu	Glu	Ala	Gly	Thr	Pro	Ala	Ala	Gly	Glu	Lys	Gln	Leu	Ala	Met
145					150					155					160
Cys	Leu	Gln	Arg	Leu	Glu	Lys	Thr	Leu	Ser	Ser	Thr	Lys	Asn	Arg	Ala
				165					170					175	
Leu	Leu	His	Ile	Ala	Lys	Leu	Glu	Glu	Ala	Ser	Ser	Trp	Thr	Ala	Ile
			180					185					190		
Glu	His	Ser	Leu	Leu	Lys	Leu	Gly	Glu	Ile	Leu	Thr	Asn	Leu	Ser	Asn
		195					200					205			
Pro	Gln	Leu	Arg	Ser	Gln	Ala	Glu	Gln	Cys	Gly	Thr	Leu	Ile	Arg	Ser
	210					215					220				
Ile	Pro	Thr	Met	Leu	Ser	Val	His	Ala	Glu	Gln	Met	His	Lys	Thr	Gly
225					230					235					240
Phe	Pro	Thr	Val	His	Ala	Val	Ile	Leu	Leu	Glu	Gly	Thr	Met	Asn	Leu
				245					250					255	
Thr	Gly	Glu	Thr	Gln	Ser	Leu	Val	Glu	Gln	Leu	Thr	Met	Val	Lys	Arg
			260					265					270		
Met	Gln	His	Ile	Pro	Thr	Pro	Leu	Phe	Val	Leu	Glu	Ile	Trp	Lys	Ala
		275					280					285			
Cys	Phe	Val	Gly	Leu	Ile	Glu	Ser	Pro	Glu	Gly	Thr	Glu	Glu	Leu	Lys
	290					295					300				
Trp	Thr	Ala	Phe	Thr	Phe	Leu	Lys	Ile	Pro	Gln	Val	Leu	Val	Lys	Leu
305					310					315					320
Lys	Lys	Tyr	Ser	His	Gly	Asp	Lys	Asp	Phe	Thr	Glu	Asp	Val	Asn	Cys
				325					330					335	
Ala	Phe	Glu	Phe	Leu	Leu	Lys	Leu	Thr	Pro	Leu	Leu	Asp	Lys	Ala	Asp
			340					345					350		
Gln	Arg	Cys	Asn	Cys	Asp	Cys	Thr	Asn	Phe	Leu	Leu	Gln	Glu	Cys	Gly
		355					360					365			
Lys	Gln	Gly	Leu	Leu	Ser	Glu	Ala	Ser	Val	Asn	Asn	Leu	Met	Ala	Lys
	370					375					380				
Arg	Lys	Ala	Asp	Arg	Glu	His	Ala	Pro	Gln	Gln	Lys	Ser	Gly	Glu	Asn
385					390					395					400
Ala	Asn	Ile	Gln	Pro	Asn	Ile	Gln	Leu	Ile	Leu	Arg	Ala	Glu	Pro	Thr
				405					410					415	
Val	Thr	Asn	Ile	Leu	Lys	Thr	Met	Asp	Ala	Asp	His	Ser	Lys	Ser	Pro
		420						425					430		
Glu	Gly	Leu	Leu	Gly	Val	Leu	Gly	His	Met	Leu	Ser	Gly	Lys	Ser	Leu
		435					440					445			
Asp	Leu	Leu	Leu	Ala	Ala	Ala	Ala	Ala	Thr	Gly	Lys	Leu	Lys	Ser	Phe
	450					455					460				
Ala	Arg	Lys	Phe	Ile	Asn	Leu	Asn	Glu	Phe	Thr	Thr	Tyr	Gly	Ser	Glu
465					470					475					480
Glu	Ser	Thr	Lys	Pro	Ala	Ser	Val	Arg	Ala	Leu	Leu	Phe	Asp	Ile	Ser
				485					490					495	
Phe	Leu	Met	Leu	Cys	His	Val	Ala	Gln	Thr	Tyr	Gly	Ser	Glu	Val	Ile
		500						505					510		
Leu	Ser	Glu	Ser	Arg	Thr	Gly	Ala	Glu	Val	Pro	Phe	Phe	Glu	Thr	Trp
		515					520					525			
Met	Gln	Thr	Cys	Met	Pro	Glu	Glu	Gly	Lys	Ile	Leu	Asn	Pro	Asp	His
	530					535					540				
Pro	Cys	Phe	Arg	Pro	Asp	Ser	Thr	Lys	Val	Glu	Ser	Leu	Val	Ala	Leu
545					550					555					560
Leu	Asn	Asn	Ser	Ser	Glu	Met	Lys	Leu	Val	Gln	Met	Lys	Trp	His	Glu
				565					570					575	
Ala	Cys	Leu	Ser	Ile	Ser	Ala	Ala	Ile	Leu	Glu	Ile	Leu	Asn	Ala	Trp
			580					585					590		
Glu	Asn	Gly	Val	Leu	Ala	Phe	Glu	Ser	Ile	Gln	Lys	Ile	Thr	Asp	Asn
		595					600						605		

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Ile Lys Gly Lys Val Cys Ser Leu Ala Val Cys Ala Val Ala Trp Leu
 610          615          620
Val Ala His Val Arg Met Leu Gly Leu Asp Glu Arg Glu Lys Ser Leu
625          630          635
Gln Met Ile Arg Gln Leu Ala Gly Pro Leu Phe Ser Glu Asn Thr Leu
          645          650          655
Gln Phe Tyr Asn Glu Arg Val Val Ile Met Asn Ser Ile Leu Glu Arg
          660          665          670
Met Cys Ala Asp Val Leu Gln Gln Thr Ala Thr Gln Ile Lys Phe Pro
          675          680          685
Ser Thr Gly Val Asp Thr Met Pro Tyr Trp Asn Leu Leu Pro Pro Lys
          690          695          700
Arg Pro Ile Lys Glu Val Leu Thr Asp Ile Phe Ala Lys Val Leu Glu
705          710          715          720
Lys Gly Trp Val Asp Ser Arg Ser Ile His Ile Phe Asp Thr Leu Leu
          725          730          735
His Met Gly Gly Val Tyr Trp Phe Cys Asn Asn Leu Ile Lys Glu Leu
          740          745          750
Leu Lys Glu Thr Arg Lys Glu His Thr Leu Arg Ala Val Glu Leu Leu
          755          760          765
Tyr Ser Ile Phe Cys Leu Asp Met Gln Gln Val Thr Leu Val Leu Leu
          770          775          780
Gly His Ile Leu Pro Gly Leu Leu Thr Asp Ser Ser Lys Trp His Ser
785          790          795          800
Leu Met Asp Pro Pro Gly Thr Ala Leu Ala Lys Leu Ala Val Trp Cys
          805          810          815
Ala Leu Ser Ser Tyr Ser Ser His Lys Gly Gln Ala Ser Thr Arg Gln
          820          825          830
Lys Lys Arg His Arg Glu Asp Ile Glu Asp Tyr Ile Ser Leu Phe Pro
          835          840          845
Leu Asp Asp Val Gln Pro Ser Lys Leu Met Arg Leu Leu Ser Ser Asn
          850          855          860
Glu Asp Asp Ala Asn Ile Leu Ser Ser Pro Thr Asp Arg Ser Met Ser
865          870          875          880
Ser Ser Leu Ser Ala Ser Gln Leu His Thr Val Asn Met Arg Asp Pro
          885          890          895
Leu Asn Arg Val Leu Ala Asn Leu Phe Leu Leu Ile Ser Ser Ile Leu
          900          905          910
Gly Ser Arg Thr Ala Gly Pro His Thr Gln Phe Val Gln Trp Phe Met
          915          920          925
Glu Glu Cys Val Asp Cys Leu Glu Gln Gly Gly Arg Gly Ser Val Leu
          930          935          940
Gln Phe Met Pro Phe Thr Thr Val Ser Glu Leu Val Lys Val Ser Ala
945          950          955          960
Met Ser Ser Pro Lys Val Val Leu Ala Ile Thr Asp Leu Ser Leu Pro
          965          970          975
Leu Gly Arg Gln Val Ala Ala Lys Ala Ile Ala Ala Leu
          980          985

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<210> 41

<211> 490

<212> PRT

<213> Homo sapiens

<400> 41

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Met Glu Gln Lys Pro Ser Lys Val Glu Cys Gly Ser Asp Pro Glu Glu
 1          5          10          15
Asn Ser Ala Arg Ser Pro Asp Gly Lys Arg Lys Arg Lys Asn Gly Gln
          20          25          30
Cys Ser Leu Lys Thr Ser Met Ser Gly Tyr Ile Pro Ser Tyr Leu Asp
          35          40          45

```

Lys Asp Glu Gln Cys Val Val Cys Gly Asp Lys Ala Thr Gly Tyr His
 50 55 60
 Tyr Arg Cys Ile Thr Cys Glu Gly Cys Lys Gly Phe Phe Arg Arg Thr
 65 70 75 80
 Ile Gln Lys Asn Leu His Pro Thr Tyr Ser Cys Lys Tyr Asp Ser Cys
 85 90 95
 Cys Val Ile Asp Lys Ile Thr Arg Asn Gln Cys Gln Leu Cys Arg Phe
 100 105 110
 Lys Lys Cys Ile Ala Val Gly Met Ala Met Asp Leu Val Leu Asp Asp
 115 120 125
 Ser Lys Arg Val Ala Lys Arg Lys Leu Ile Glu Gln Asn Arg Glu Arg
 130 135 140
 Arg Arg Lys Glu Glu Met Ile Arg Ser Leu Gln Arg Pro Glu Pro
 145 150 155 160
 Thr Pro Glu Glu Trp Asp Leu Ile His Ile Ala Thr Glu Ala His Arg
 165 170 175
 Ser Thr Asn Ala Gln Gly Ser His Trp Lys Gln Arg Arg Lys Phe Leu
 180 185 190
 Pro Asp Asp Ile Gly Gln Ser Pro Ile Val Ser Met Pro Asp Gly Asp
 195 200 205
 Lys Val Asp Leu Glu Ala Phe Ser Glu Phe Thr Lys Ile Ile Thr Pro
 210 215 220
 Ala Ile Thr Arg Val Val Asp Phe Ala Lys Lys Leu Pro Met Phe Ser
 225 230 235 240
 Glu Leu Pro Cys Glu Asp Gln Ile Ile Leu Leu Lys Gly Cys Cys Met
 245 250 255
 Glu Ile Met Ser Leu Arg Ala Ala Val Arg Tyr Asp Pro Glu Ser Asp
 260 265 270
 Thr Leu Thr Leu Ser Gly Glu Met Ala Val Lys Arg Glu Gln Leu Lys
 275 280 285
 Asn Gly Gly Leu Gly Val Val Ser Asp Ala Ile Phe Glu Leu Gly Lys
 290 295 300
 Ser Leu Ser Ala Phe Asn Leu Asp Asp Thr Glu Val Ala Leu Leu Gln
 305 310 315 320
 Ala Val Leu Leu Met Ser Thr Asp Arg Ser Gly Leu Leu Cys Val Asp
 325 330 335
 Lys Ile Glu Lys Ser Gln Glu Ala Tyr Leu Leu Ala Phe Glu His Tyr
 340 345 350
 Val Asn His Arg Lys His Asn Ile Pro His Phe Trp Pro Lys Leu Leu
 355 360 365
 Met Lys Glu Arg Glu Val Gln Ser Ser Ile Leu Tyr Lys Gly Ala Ala
 370 375 380
 Ala Glu Gly Arg Pro Gly Gly Ser Leu Gly Val His Pro Glu Gly Gln
 385 390 395 400
 Gln Leu Leu Gly Met His Val Val Gln Gly Pro Gln Val Arg Gln Leu
 405 410 415
 Glu Gln Gln Leu Gly Glu Ala Gly Ser Leu Gln Gly Pro Val Leu Gln
 420 425 430
 His Gln Ser Pro Lys Ser Pro Gln Gln Arg Leu Leu Glu Leu Leu His
 435 440 445
 Arg Ser Gly Ile Leu His Ala Arg Ala Val Cys Gly Glu Asp Asp Ser
 450 455 460
 Ser Glu Ala Asp Ser Pro Ser Ser Ser Glu Glu Pro Glu Val Cys
 465 470 475 480
 Glu Asp Leu Ala Gly Asn Ala Ala Ser Pro
 485 490

<210> 42

<211> 614

<212> PRT

<213> Homo sapiens

<400> 42

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Met Thr Thr Leu Asp Ser Asn Asn Asn Thr Gly Gly Val Ile Thr Tyr
1      5      10      15
Ile Gly Ser Ser Gly Ser Ser Pro Ser Arg Thr Ser Pro Glu Ser Leu
20      25      30
Tyr Ser Asp Asn Ser Asn Gly Ser Phe Gln Ser Leu Thr Gln Gly Cys
35      40      45
Pro Thr Tyr Phe Pro Pro Ser Pro Thr Gly Ser Leu Thr Gln Asp Pro
50      55      60
Ala Arg Ser Phe Gly Ser Ile Pro Pro Ser Leu Ser Asp Asp Gly Ser
65      70      75      80
Pro Ser Ser Ser Ser Ser Ser Ser Ser Ser Ser Ser Ser Phe Tyr Asn
85      90      95
Gly Ser Pro Pro Gly Ser Leu Gln Val Ala Met Glu Asp Ser Ser Arg
100     105     110
Val Ser Pro Ser Lys Ser Thr Ser Asn Ile Thr Lys Leu Asn Gly Met
115     120     125
Val Leu Leu Cys Lys Val Cys Gly Asp Val Ala Ser Gly Phe His Tyr
130     135     140
Gly Val Leu Ala Cys Glu Gly Cys Lys Gly Phe Phe Arg Arg Ser Ile
145     150     155     160
Gln Gln Asn Ile Gln Tyr Lys Arg Cys Leu Lys Asn Glu Asn Cys Ser
165     170     175
Ile Val Arg Ile Asn Arg Asn Arg Cys Gln Gln Cys Arg Phe Lys Lys
180     185     190
Cys Leu Ser Val Gly Met Ser Arg Asp Ala Val Arg Phe Gly Arg Ile
195     200     205
Pro Lys Arg Glu Lys Gln Arg Met Leu Ala Glu Met Gln Ser Ala Met
210     215     220
Asn Leu Ala Asn Asn Gln Leu Ser Ser Gln Cys Pro Leu Glu Thr Ser
225     230     235     240
Pro Thr Gln His Pro Thr Pro Gly Pro Met Gly Pro Ser Pro Pro Pro
245     250     255
Ala Pro Val Pro Ser Pro Leu Val Gly Phe Ser Gln Phe Pro Gln Gln
260     265     270
Leu Thr Pro Pro Arg Ser Pro Ser Pro Glu Pro Thr Val Glu Asp Val
275     280     285
Ile Ser Gln Val Ala Arg Ala His Arg Glu Ile Phe Thr Tyr Ala His
290     295     300
Asp Lys Leu Gly Ser Ser Pro Gly Asn Phe Asn Ala Asn His Ala Ser
305     310     315     320
Gly Ser Pro Pro Ala Thr Thr Pro His Arg Trp Glu Asn Gln Gly Cys
325     330     335
Pro Pro Ala Pro Asn Asp Asn Asn Thr Leu Ala Ala Gln Arg His Asn
340     345     350
Glu Ala Leu Asn Gly Leu Arg Gln Ala Pro Ser Ser Tyr Pro Pro Thr
355     360     365
Trp Pro Pro Gly Pro Ala His Ser Cys His Gln Ser Asn Ser Asn
370     375     380
Gly His Arg Leu Cys Pro Thr His Val Tyr Ala Ala Pro Glu Gly Lys
385     390     395     400
Ala Pro Ala Asn Ser Pro Arg Gln Gly Asn Ser Lys Asn Val Leu Leu
405     410     415
Ala Cys Pro Met Asn Met Tyr Pro His Gly Arg Ser Gly Arg Thr Val
420     425     430
Gln Glu Ile Trp Glu Asp Phe Ser Met Ser Phe Thr Pro Ala Val Arg
435     440     445
Glu Val Val Glu Phe Ala Lys His Ile Pro Gly Phe Arg Asp Leu Ser
450     455     460
Gln His Asp Gln Val Thr Leu Leu Lys Ala Gly Thr Phe Glu Val Leu
465     470     475     480
Met Val Arg Phe Ala Ser Leu Phe Asn Val Lys Asp Gln Thr Val Met
485     490     495
Phe Leu Ser Arg Thr Thr Tyr Ser Leu Gln Glu Leu Gly Ala Met Gly
500     505     510

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Met. Gly Asp Leu Leu Ser Ala Met Phe Asp Phe Ser Glu Lys Leu Asn
 515 520 525
 Ser Leu Ala Leu Thr Glu Glu Glu Leu Gly Leu Phe Thr Ala Val Val
 530 535 540
 Leu Val Ser Ala Asp Arg Ser Gly Met Glu Asn Ser Ala Ser Val Glu
 545 550 555 560
 Gln Leu Gln Glu Thr Leu Leu Arg Ala Leu Arg Ala Leu Val Leu Lys
 565 570 575
 Asn Arg Pro Leu Glu Thr Ser Arg Phe Thr Lys Leu Leu Leu Lys Leu
 580 585 590
 Pro Asp Leu Arg Thr Leu Asn Asn Met His Ser Glu Lys Leu Leu Ser
 595 600 605
 Phe Arg Val Asp Ala Gln
 610

<210> 43

<211> 703

<212> PRT

<213> Homo sapiens

<400> 43

Met Ala Asp Arg Arg Arg Gln Arg Ala Ser Gln Asp Thr Glu Asp Glu
 1 5 10 15
 Glu Ser Gly Ala Ser Gly Ser Asp Ser Gly Gly Ser Pro Leu Arg Gly
 20 25 30
 Gly Gly Ser Cys Ser Gly Ser Ala Gly Gly Gly Gly Ser Gly Ser Leu
 35 40 45
 Pro Ser Gln Arg Gly Gly Arg Thr Gly Ala Leu His Leu Arg Arg Val
 50 55 60
 Glu Ser Gly Gly Ala Lys Ser Ala Glu Glu Ser Glu Cys Glu Ser Glu
 65 70 75 80
 Asp Gly Ile Glu Gly Asp Ala Val Leu Ser Asp Tyr Glu Ser Ala Glu
 85 90 95
 Asp Ser Glu Gly Glu Glu Gly Glu Tyr Ser Glu Glu Glu Asn Ser Lys
 100 105 110
 Val Glu Leu Lys Ser Glu Ala Asn Asp Ala Val Asn Ser Ser Thr Lys
 115 120 125
 Glu Glu Lys Gly Glu Glu Lys Pro Asp Thr Lys Ser Thr Val Thr Gly
 130 135 140
 Glu Arg Gln Ser Gly Asp Gly Gln Glu Ser Thr Glu Pro Val Glu Asn
 145 150 155 160
 Lys Val Gly Lys Lys Gly Pro Lys His Leu Asp Asp Asp Glu Asp Arg
 165 170 175
 Lys Asn Pro Ala Tyr Ile Pro Arg Lys Gly Leu Phe Phe Glu His Asp
 180 185 190
 Leu Arg Gly Gln Thr Gln Glu Glu Val Arg Pro Lys Gly Arg Gln
 195 200 205
 Arg Lys Leu Trp Lys Asp Glu Gly Arg Trp Glu His Asp Lys Phe Arg
 210 215 220
 Glu Asp Glu Gln Ala Pro Lys Ser Arg Gln Glu Leu Ile Ala Leu Tyr
 225 230 235 240
 Gly Tyr Asp Ile Arg Ser Ala His Asn Pro Asp Asp Ile Lys Pro Arg
 245 250 255
 Arg Ile Arg Lys Pro Arg Tyr Gly Ser Pro Pro Gln Arg Asp Pro Asn
 260 265 270
 Trp Asn Gly Glu Arg Leu Asn Lys Ser His Arg His Gln Gly Leu Gly
 275 280 285
 Gly Thr Leu Pro Pro Arg Thr Phe Ile Asn Arg Asn Ala Ala Gly Thr
 290 295 300
 Gly Arg Met Ser Ala Pro Arg Asn Tyr Ser Arg Ser Gly Gly Phe Lys
 305 310 315 320

Glu Gly Arg Ala Gly Phe Arg Pro Val Glu Ala Gly Gly Gln His Gly
 325 330 335
 Gly Arg Ser Gly Glu Thr Val Lys His Glu Ile Ser Tyr Arg Ser Arg
 340 345 350
 Arg Leu Glu Gln Thr Ser Val Arg Asp Pro Ser Pro Glu Ala Asp Ala
 355 360 365
 Pro Val Leu Gly Ser Pro Glu Lys Glu Glu Ala Ala Ser Glu Pro Pro
 370 375 380
 Ala Ala Ala Pro Asp Ala Ala Pro Pro Pro Pro Asp Arg Pro Ile Glu
 385 390 395 400
 Lys Lys Ser Tyr Ser Arg Ala Arg Arg Thr Arg Thr Lys Val Gly Asp
 405 410 415
 Ala Val Lys Leu Ala Glu Glu Val Pro Pro Pro Pro Glu Gly Leu Ile
 420 425 430
 Pro Ala Pro Pro Val Pro Glu Thr Thr Pro Thr Pro Pro Thr Lys Thr
 435 440 445
 Gly Thr Trp Glu Ala Pro Val Asp Ser Ser Thr Ser Gly Leu Glu Gln
 450 455 460
 Asp Val Ala Gln Leu Asn Ile Ala Glu Gln Asn Trp Ser Pro Gly Gln
 465 470 475 480
 Pro Ser Phe Leu Gln Pro Arg Glu Leu Arg Gly Met Pro Asn His Ile
 485 490 495
 His Met Gly Ala Gly Pro Pro Pro Gln Phe Asn Arg Met Glu Glu Met
 500 505 510
 Gly Val Gln Gly Gly Arg Ala Lys Arg Tyr Ser Ser Gln Arg Gln Arg
 515 520 525
 Pro Val Pro Glu Pro Pro Ala Pro Pro Val His Ile Ser Ile Met Glu
 530 535 540
 Gly His Tyr Tyr Asp Pro Leu Gln Phe Gln Gly Pro Ile Tyr Thr His
 545 550 555 560
 Gly Asp Ser Pro Ala Pro Leu Pro Pro Gln Gly Met Leu Val Gln Pro
 565 570 575
 Gly Met Asn Leu Pro His Pro Gly Leu His Pro His Gln Thr Pro Ala
 580 585 590
 Pro Leu Pro Asn Pro Gly Leu Tyr Pro Pro Pro Val Ser Met Ser Pro
 595 600 605
 Gly Gln Pro Pro Pro Gln Gln Leu Leu Ala Pro Thr Tyr Phe Ser Ala
 610 615 620
 Pro Gly Val Met Asn Phe Gly Asn Pro Ser Tyr Pro Tyr Ala Pro Gly
 625 630 635 640
 Ala Leu Pro Pro Pro Pro Pro Pro His Leu Tyr Pro Asn Thr Gln Ala
 645 650 655
 Pro Ser Gln Val Tyr Gly Gly Val Thr Tyr Tyr Asn Pro Ala Gln Gln
 660 665 670
 Gln Val Gln Pro Lys Pro Ser Pro Pro Arg Arg Thr Pro Gln Pro Val
 675 680 685
 Thr Ile Lys Pro Pro Pro Pro Glu Val Val Ser Arg Gly Ser Ser
 690 695 700

<210> 44

<211> 560

<212> PRT

<213> Homo sapiens

<400> 44

Met Pro Gln Thr Arg Ser Gln Ala Gln Ala Thr Ile Ser Phe Pro Lys
 1 5 10 15
 Arg Lys Leu Ser Arg Ala Leu Asn Lys Ala Lys Asn Ser Ser Asp Ala
 20 25 30
 Lys Leu Glu Pro Thr Asn Val Gln Thr Val Thr Cys Ser Pro Arg Val
 35 40 45

Lys Ala Leu Pro Leu Ser Pro Arg Lys Arg Leu Gly Asp Asp Asn Leu
 50 55 60
 Cys Asn Thr Pro His Leu Pro Pro Cys Ser Pro Pro Lys Gln Gly Lys
 65 70 75 80
 Lys Glu Asn Gly Pro Pro His Ser His Thr Leu Lys Gly Arg Arg Leu
 85 90 95
 Val Phe Asp Asn Gln Leu Thr Ile Lys Ser Pro Ser Lys Arg Glu Leu
 100 105 110
 Ala Lys Val His Gln Asn Lys Ile Leu Ser Ser Val Arg Lys Ser Gln
 115 120 125
 Glu Ile Thr Thr Asn Ser Glu Gln Arg Cys Pro Leu Lys Lys Glu Ser
 130 135 140
 Ala Cys Val Arg Leu Phe Lys Gln Glu Gly Thr Cys Tyr Gln Gln Ala
 145 150 155 160
 Lys Leu Val Leu Asn Thr Ala Val Pro Asp Arg Leu Pro Ala Arg Glu
 165 170 175
 Arg Glu Met Asp Val Ile Arg Asn Phe Leu Arg Glu His Ile Cys Gly
 180 185 190
 Lys Lys Ala Gly Ser Leu Tyr Leu Ser Gly Ala Pro Gly Thr Gly Lys
 195 200 205
 Thr Ala Cys Leu Ser Arg Ile Leu Gln Asp Leu Lys Lys Glu Leu Lys
 210 215 220
 Gly Phe Lys Thr Ile Met Leu Asn Cys Met Ser Leu Arg Thr Ala Gln
 225 230 235 240
 Ala Val Phe Pro Ala Ile Ala Gln Glu Ile Cys Gln Glu Glu Val Ser
 245 250 255
 Arg Pro Ala Gly Lys Asp Met Met Arg Lys Leu Glu Lys His Met Thr
 260 265 270
 Ala Glu Lys Gly Pro Met Ile Val Leu Val Leu Asp Glu Met Asp Gln
 275 280 285
 Leu Asp Ser Lys Gly Gln Asp Val Leu Tyr Thr Leu Phe Glu Trp Pro
 290 295 300
 Trp Leu Ser Asn Ser His Leu Val Leu Ile Gly Ile Ala Asn Thr Leu
 305 310 315 320
 Asp Leu Thr Asp Arg Ile Leu Pro Arg Leu Gln Ala Arg Glu Lys Cys
 325 330 335
 Lys Pro Gln Leu Leu Asn Phe Pro Pro Tyr Thr Arg Asn Gln Ile Val
 340 345 350
 Thr Ile Leu Gln Asp Arg Leu Asn Gln Val Ser Arg Asp Gln Val Leu
 355 360 365
 Asp Asn Ala Ala Val Gln Phe Cys Ala Arg Lys Val Ser Ala Val Ser
 370 375 380
 Gly Asp Val Arg Lys Ala Leu Asp Val Cys Arg Arg Ala Ile Glu Ile
 385 390 395 400
 Val Glu Ser Asp Val Lys Ser Gln Thr Ile Leu Lys Pro Leu Ser Glu
 405 410 415
 Cys Lys Ser Pro Ser Glu Pro Leu Ile Pro Lys Arg Val Gly Leu Ile
 420 425 430
 His Ile Ser Gln Val Ile Ser Glu Val Asp Gly Asn Arg Met Thr Leu
 435 440 445
 Ser Gln Glu Gly Ala Gln Asp Ser Phe Pro Leu Gln Gln Lys Ile Leu
 450 455 460
 Val Cys Ser Leu Met Leu Leu Ile Arg Gln Leu Lys Ile Lys Glu Val
 465 470 475 480
 Thr Leu Gly Lys Leu Tyr Glu Ala Tyr Ser Lys Val Cys Arg Lys Gln
 485 490 495
 Gln Val Ala Ala Val Asp Gln Ser Glu Cys Leu Ser Leu Ser Gly Leu
 500 505 510
 Leu Glu Ala Arg Gly Ile Leu Gly Leu Lys Arg Asn Lys Glu Thr Arg
 515 520 525
 Leu Thr Lys Val Phe Phe Lys Ile Glu Glu Lys Glu Ile Glu His Ala
 530 535 540
 Leu Lys Asp Lys Ala Leu Ile Gly Asn Ile Leu Ala Thr Gly Leu Pro
 545 550 555 560
 <210> 45

<211> 462

<212> PRT

<213> Homo sapiens

<400> 45

Met	Ala	Ser	Asn	Ser	Ser	Ser	Cys	Pro	Thr	Pro	Gly	Gly	Gly	His	Leu
1				5					10					15	
Asn	Gly	Tyr	Pro	Val	Pro	Pro	Tyr	Ala	Phe	Phe	Phe	Pro	Pro	Met	Leu
			20					25					30		
Gly	Gly	Leu	Ser	Pro	Pro	Gly	Ala	Leu	Thr	Thr	Leu	Gln	His	Gln	Leu
		35					40					45			
Pro	Val	Ser	Gly	Tyr	Ser	Thr	Pro	Ser	Pro	Ala	Thr	Ile	Glu	Thr	Gln
	50					55					60				
Ser	Ser	Ser	Ser	Glu	Glu	Ile	Val	Pro	Ser	Pro	Pro	Ser	Pro	Pro	Pro
65				70						75					80
Leu	Pro	Arg	Ile	Tyr	Lys	Pro	Cys	Phe	Val	Cys	Gln	Asp	Lys	Ser	Ser
			85						90					95	
Gly	Tyr	His	Tyr	Gly	Val	Ser	Ala	Cys	Glu	Gly	Cys	Lys	Gly	Phe	Phe
			100					105					110		
Arg	Arg	Ser	Ile	Gln	Lys	Asn	Met	Val	Tyr	Thr	Cys	His	Arg	Asp	Lys
		115					120					125			
Asn	Cys	Ile	Ile	Asn	Lys	Val	Thr	Arg	Asn	Arg	Cys	Gln	Tyr	Cys	Arg
	130					135					140				
Leu	Gln	Lys	Cys	Phe	Glu	Val	Gly	Met	Ser	Lys	Glu	Ser	Val	Arg	Asn
145					150					155					160
Asp	Arg	Asn	Lys	Lys	Lys	Lys	Glu	Val	Pro	Lys	Pro	Glu	Cys	Ser	Glu
			165						170					175	
Ser	Tyr	Thr	Leu	Thr	Pro	Glu	Val	Gly	Glu	Leu	Ile	Glu	Lys	Val	Arg
			180					185					190		
Lys	Ala	His	Gln	Glu	Thr	Phe	Pro	Ala	Leu	Cys	Gln	Leu	Gly	Lys	Tyr
		195					200					205			
Thr	Thr	Asn	Asn	Ser	Ser	Glu	Gln	Arg	Val	Ser	Leu	Asp	Ile	Asp	Leu
	210					215					220				
Trp	Asp	Lys	Phe	Ser	Glu	Leu	Ser	Thr	Lys	Cys	Ile	Ile	Lys	Thr	Val
225					230					235					240
Glu	Phe	Ala	Lys	Gln	Leu	Pro	Gly	Phe	Thr	Thr	Leu	Thr	Ile	Ala	Asp
			245						250					255	
Gln	Ile	Thr	Leu	Leu	Lys	Ala	Ala	Cys	Leu	Asp	Ile	Leu	Ile	Leu	Arg
			260					265					270		
Ile	Cys	Thr	Arg	Tyr	Thr	Pro	Glu	Gln	Asp	Thr	Met	Thr	Phe	Ser	Asp
		275					280					285			
Gly	Leu	Thr	Leu	Asn	Arg	Thr	Gln	Met	His	Asn	Ala	Gly	Phe	Gly	Pro
	290					295					300				
Leu	Thr	Asp	Leu	Val	Phe	Ala	Phe	Ala	Asn	Gln	Leu	Leu	Pro	Leu	Glu
305					310					315					320
Met	Asp	Asp	Ala	Glu	Thr	Gly	Leu	Leu	Ser	Ala	Ile	Cys	Leu	Ile	Cys
			325						330					335	
Gly	Asp	Arg	Gln	Asp	Leu	Glu	Gln	Pro	Asp	Arg	Val	Asp	Met	Leu	Gln
			340					345					350		
Glu	Pro	Leu	Leu	Glu	Ala	Leu	Lys	Val	Tyr	Val	Arg	Lys	Arg	Arg	Pro
		355					360					365			
Ser	Arg	Pro	His	Met	Phe	Pro	Lys	Met	Leu	Met	Lys	Ile	Thr	Asp	Leu
	370					375					380				
Arg	Ser	Ile	Ser	Ala	Lys	Gly	Ala	Glu	Arg	Val	Ile	Thr	Leu	Lys	Met
385					390					395					400
Glu	Ile	Pro	Gly	Ser	Met	Pro	Pro	Leu	Ile	Gln	Glu	Met	Leu	Glu	Asn
			405						410					415	
Ser	Glu	Gly	Leu	Asp	Thr	Leu	Ser	Gly	Gln	Pro	Gly	Gly	Gly	Gly	Arg
			420					425					430		
Asp	Gly	Gly	Gly	Leu	Ala	Pro	Pro	Pro	Gly	Ser	Cys	Ser	Pro	Ser	Leu
		435					440					445			

Ser Pro Ser Ser Asn Arg Ser Ser Pro Ala Thr His Ser Pro
 450 455 460

<210> 46

<211> 1531

<212> PRT

<213> Homo sapiens

<400> 46

Met	Glu	Val	Ser	Pro	Leu	Gln	Pro	Val	Asn	Glu	Asn	Met	Gln	Val	Asn
1				5					10					15	
Lys	Ile	Lys	Lys	Asn	Glu	Asp	Ala	Lys	Lys	Arg	Leu	Ser	Val	Glu	Arg
			20					25					30		
Ile	Tyr	Gln	Lys	Lys	Thr	Gln	Leu	Glu	His	Ile	Leu	Leu	Arg	Pro	Asp
		35					40					45			
Thr	Tyr	Ile	Gly	Ser	Val	Glu	Leu	Val	Thr	Gln	Gln	Met	Trp	Val	Tyr
		50				55					60				
Asp	Glu	Asp	Val	Gly	Ile	Asn	Tyr	Arg	Glu	Val	Thr	Phe	Val	Pro	Gly
65				70					75					80	
Leu	Tyr	Lys	Ile	Phe	Asp	Glu	Ile	Leu	Val	Asn	Ala	Ala	Asp	Asn	Lys
				85					90					95	
Gln	Arg	Asp	Pro	Lys	Met	Ser	Cys	Ile	Arg	Val	Thr	Ile	Asp	Pro	Glu
			100					105					110		
Asn	Asn	Leu	Ile	Ser	Ile	Trp	Asn	Asn	Gly	Lys	Gly	Ile	Pro	Val	Val
		115					120					125			
Glu	His	Lys	Val	Glu	Lys	Met	Tyr	Val	Pro	Ala	Leu	Ile	Phe	Gly	Gln
	130					135					140				
Leu	Leu	Thr	Ser	Ser	Asn	Tyr	Asp	Asp	Asp	Glu	Lys	Lys	Val	Thr	Gly
145					150					155					160
Gly	Arg	Asn	Gly	Tyr	Gly	Ala	Lys	Leu	Cys	Asn	Ile	Phe	Ser	Thr	Lys
				165					170					175	
Phe	Thr	Val	Glu	Thr	Ala	Ser	Arg	Glu	Tyr	Lys	Lys	Met	Phe	Lys	Gln
			180					185					190		
Thr	Trp	Met	Asp	Asn	Met	Gly	Arg	Ala	Gly	Glu	Met	Glu	Leu	Lys	Pro
		195					200					205			
Phe	Asn	Gly	Glu	Asp	Tyr	Thr	Cys	Ile	Thr	Phe	Gln	Pro	Asp	Leu	Ser
	210					215					220				
Lys	Phe	Lys	Met	Gln	Ser	Leu	Asp	Lys	Asp	Ile	Val	Ala	Leu	Met	Val
225					230					235					240
Arg	Arg	Ala	Tyr	Asp	Ile	Ala	Gly	Ser	Thr	Lys	Asp	Val	Lys	Val	Phe
				245					250					255	
Leu	Asn	Gly	Asn	Lys	Leu	Pro	Val	Lys	Gly	Phe	Arg	Ser	Tyr	Val	Asp
			260					265					270		
Met	Tyr	Leu	Lys	Asp	Lys	Leu	Asp	Glu	Thr	Gly	Asn	Ser	Leu	Lys	Val
		275					280					285			
Ile	His	Glu	Gln	Val	Asn	His	Arg	Trp	Glu	Val	Cys	Leu	Thr	Met	Ser
	290					295					300				
Glu	Lys	Gly	Phe	Gln	Gln	Ile	Ser	Phe	Val	Asn	Ser	Ile	Ala	Thr	Ser
305					310					315					320
Lys	Gly	Gly	Arg	His	Val	Asp	Tyr	Val	Ala	Asp	Gln	Ile	Val	Thr	Lys
				325					330					335	
Leu	Val	Asp	Val	Val	Lys	Lys	Lys	Asn	Lys	Gly	Gly	Val	Ala	Val	Lys
			340					345					350		
Ala	His	Gln	Val	Lys	Asn	His	Met	Trp	Ile	Phe	Val	Asn	Ala	Leu	Ile
	355						360					365			
Glu	Asn	Pro	Thr	Phe	Asp	Ser	Gln	Thr	Lys	Glu	Asn	Met	Thr	Leu	Gln
	370					375					380				
Pro	Lys	Ser	Phe	Gly	Ser	Thr	Cys	Gln	Leu	Ser	Glu	Lys	Phe	Ile	Lys
385					390					395					400
Ala	Ala	Ile	Gly	Cys	Gly	Ile	Val	Glu	Ser	Ile	Leu	Asn	Trp	Val	Lys
				405					410					415	

Phe	Lys	Ala	Gln	Val	Gln	Leu	Asn	Lys	Lys	Cys	Ser	Ala	Val	Lys	His
Asn	Arg	Ile	Lys	Gly	Ile	Pro	Lys	Leu	Asp	Asp	Ala	Asn	Asp	Ala	Gly
Gly	Arg	Asn	Ser	Thr	Glu	Cys	Thr	Leu	Ile	Leu	Thr	Glu	Gly	Asp	Ser
Ala	Lys	Thr	Leu	Ala	Val	Ser	Gly	Leu	Gly	Val	Val	Gly	Arg	Asp	Lys
Tyr	Gly	Val	Phe	Pro	Leu	Arg	Gly	Lys	Ile	Leu	Asn	Val	Arg	Glu	Ala
Ser	His	Lys	Gln	Ile	Met	Glu	Asn	Ala	Glu	Ile	Asn	Asn	Ile	Ile	Lys
Ile	Val	Gly	Leu	Gln	Tyr	Lys	Lys	Asn	Tyr	Glu	Asp	Glu	Asp	Ser	Leu
Lys	Thr	Leu	Arg	Tyr	Gly	Lys	Ile	Met	Ile	Met	Thr	Asp	Gln	Asp	Gln
Asp	Gly	Ser	His	Ile	Lys	Gly	Leu	Leu	Ile	Asn	Phe	Ile	His	His	Asn
Trp	Pro	Ser	Leu	Leu	Arg	His	Arg	Phe	Leu	Glu	Glu	Phe	Ile	Thr	Pro
Ile	Val	Lys	Val	Ser	Lys	Asn	Lys	Gln	Glu	Met	Ala	Phe	Tyr	Ser	Leu
Pro	Glu	Phe	Glu	Glu	Trp	Lys	Ser	Ser	Thr	Pro	Asn	His	Lys	Lys	Trp
Lys	Val	Lys	Tyr	Tyr	Lys	Gly	Leu	Gly	Thr	Ser	Thr	Ser	Lys	Glu	Ala
Lys	Glu	Tyr	Phe	Ala	Asp	Met	Lys	Arg	His	Arg	Ile	Gln	Phe	Lys	Tyr
Ser	Gly	Pro	Glu	Asp	Asp	Ala	Ala	Ile	Ser	Leu	Ala	Phe	Ser	Lys	Lys
Gln	Ile	Asp	Asp	Arg	Lys	Glu	Trp	Leu	Thr	Asn	Phe	Met	Glu	Asp	Arg
Arg	Gln	Arg	Lys	Leu	Leu	Gly	Leu	Pro	Glu	Asp	Tyr	Leu	Tyr	Gly	Gln
Thr	Thr	Thr	Tyr	Leu	Thr	Tyr	Asn	Asp	Phe	Ile	Asn	Lys	Glu	Leu	Ile
Leu	Phe	Ser	Asn	Ser	Asp	Asn	Glu	Arg	Ser	Ile	Pro	Ser	Met	Val	Asp
Gly	Leu	Lys	Pro	Gly	Gln	Arg	Lys	Val	Leu	Phe	Thr	Cys	Phe	Lys	Arg
Asn	Asp	Lys	Arg	Glu	Val	Lys	Val	Ala	Gln	Leu	Ala	Gly	Ser	Val	Ala
Glu	Met	Ser	Ser	Tyr	His	His	Gly	Glu	Met	Ser	Leu	Met	Met	Thr	Ile
Ile	Asn	Leu	Ala	Gln	Asn	Phe	Val	Gly	Ser	Asn	Asn	Leu	Asn	Leu	Leu
Gln	Pro	Ile	Gly	Gln	Phe	Gly	Thr	Arg	Leu	His	Gly	Gly	Lys	Asp	Ser
Ala	Ser	Pro	Arg	Tyr	Ile	Phe	Thr	Met	Leu	Ser	Ser	Leu	Ala	Arg	Leu
Leu	Phe	Pro	Pro	Lys	Asp	Asp	His	Thr	Leu	Lys	Phe	Leu	Tyr	Asp	Asp
Asn	Gln	Arg	Val	Glu	Pro	Glu	Trp	Tyr	Ile	Pro	Ile	Ile	Pro	Met	Val
Leu	Ile	Asn	Gly	Ala	Glu	Gly	Ile	Gly	Thr	Gly	Trp	Ser	Cys	Lys	Ile
Pro	Asn	Phe	Asp	Val	Arg	Glu	Ile	Val	Asn	Asn	Ile	Arg	Arg	Leu	Met
Asp	Gly	Glu	Glu	Pro	Leu	Pro	Met	Leu	Pro	Ser	Tyr	Lys	Asn	Phe	Lys
Gly	Thr	Ile	Glu	Glu	Leu	Ala	Pro	Asn	Gln	Tyr	Val	Ile	Ser	Gly	Glu
Val	Ala	Ile	Leu	Asn	Ser	Thr	Thr	Ile	Glu	Ile	Ser	Glu	Leu	Pro	Val
Arg	Thr	Trp	Thr	Gln	Thr	Tyr	Lys	Glu	Gln	Val	Leu	Glu	Pro	Met	Leu

Asn	Gly	Thr	Glu	Lys	Thr	Pro	Pro	Leu	Ile	Thr	Asp	Tyr	Arg	Glu	Tyr
945					950					955					960
His	Thr	Asp	Thr	Thr	Val	Lys	Phe	Val	Val	Lys	Met	Thr	Glu	Glu	Lys
				965					970						975
Leu	Ala	Glu	Ala	Glu	Arg	Val	Gly	Leu	His	Lys	Val	Phe	Lys	Leu	Gln
			980					985					990		
Thr	Ser	Leu	Thr	Cys	Asn	Ser	Met	Val	Leu	Phe	Asp	His	Val	Gly	Cys
		995					1000					1005			
Leu	Lys	Lys	Tyr	Asp	Thr	Val	Leu	Asp	Ile	Leu	Arg	Asp	Phe	Phe	
1010						1015					1020				
Glu	Leu	Arg	Leu	Lys	Tyr	Tyr	Gly	Leu	Arg	Lys	Glu	Trp	Leu	Leu	
1025						1030					1035				
Gly	Met	Leu	Gly	Ala	Glu	Ser	Ala	Lys	Leu	Asn	Asn	Gln	Ala	Arg	
1040						1045					1050				
Phe	Ile	Leu	Glu	Lys	Ile	Asp	Gly	Lys	Ile	Ile	Ile	Glu	Asn	Lys	
1055						1060					1065				
Pro	Lys	Lys	Glu	Leu	Ile	Lys	Val	Leu	Ile	Gln	Arg	Gly	Tyr	Asp	
1070						1075					1080				
Ser	Asp	Pro	Val	Lys	Ala	Trp	Lys	Glu	Ala	Gln	Gln	Lys	Val	Pro	
1085						1090					1095				
Asp	Glu	Glu	Glu	Asn	Glu	Ser	Asp	Asn	Glu	Lys	Glu	Thr	Glu		
1100						1105					1110				
Lys	Ser	Asp	Ser	Val	Thr	Asp	Ser	Gly	Pro	Thr	Phe	Asn	Tyr	Leu	
1115						1120					1125				
Leu	Asp	Met	Pro	Leu	Trp	Tyr	Leu	Thr	Lys	Glu	Lys	Lys	Asp	Glu	
1130						1135					1140				
Leu	Cys	Arg	Leu	Arg	Asn	Glu	Lys	Glu	Gln	Glu	Leu	Asp	Thr	Leu	
1145						1150					1155				
Lys	Arg	Lys	Ser	Pro	Ser	Asp	Leu	Trp	Lys	Glu	Asp	Leu	Ala	Thr	
1160						1165					1170				
Phe	Ile	Glu	Glu	Leu	Glu	Ala	Val	Glu	Ala	Lys	Glu	Lys	Gln	Asp	
1175						1180					1185				
Glu	Gln	Val	Gly	Leu	Pro	Gly	Lys	Gly	Gly	Lys	Ala	Lys	Gly	Lys	
1190						1195					1200				
Lys	Thr	Gln	Met	Ala	Glu	Val	Leu	Pro	Ser	Pro	Arg	Gly	Gln	Arg	
1205						1210					1215				
Val	Ile	Pro	Arg	Ile	Thr	Ile	Glu	Met	Lys	Ala	Glu	Ala	Glu	Lys	
1220						1225					1230				
Lys	Asn	Lys	Lys	Lys	Ile	Lys	Asn	Glu	Asn	Thr	Glu	Gly	Ser	Pro	
1235						1240					1245				
Gln	Glu	Asp	Gly	Val	Glu	Leu	Glu	Gly	Leu	Lys	Gln	Arg	Leu	Glu	
1250						1255					1260				
Lys	Lys	Gln	Lys	Arg	Glu	Pro	Gly	Thr	Lys	Thr	Lys	Lys	Gln	Thr	
1265						1270					1275				
Thr	Leu	Ala	Phe	Lys	Pro	Ile	Lys	Lys	Gly	Lys	Lys	Arg	Asn	Pro	
1280						1285					1290				
Trp	Ser	Asp	Ser	Glu	Ser	Asp	Arg	Ser	Ser	Asp	Glu	Ser	Asn	Phe	
1295						1300					1305				
Asp	Val	Pro	Pro	Arg	Glu	Thr	Glu	Pro	Arg	Arg	Ala	Ala	Thr	Lys	
1310						1315					1320				
Thr	Lys	Phe	Thr	Met	Asp	Leu	Asp	Ser	Asp	Glu	Asp	Phe	Ser	Asp	
1325						1330					1335				
Phe	Asp	Glu	Lys	Thr	Asp	Asp	Glu	Asp	Phe	Val	Pro	Ser	Asp	Ala	
1340						1345					1350				
Ser	Pro	Pro	Lys	Thr	Lys	Thr	Ser	Pro	Lys	Leu	Ser	Asn	Lys	Glu	
1355						1360					1365				
Leu	Lys	Pro	Gln	Lys	Ser	Val	Val	Ser	Asp	Leu	Glu	Ala	Asp	Asp	
1370						1375					1380				
Val	Lys	Gly	Ser	Val	Pro	Leu	Ser	Ser	Ser	Pro	Pro	Ala	Thr	His	
1385						1390					1395				
Phe	Pro	Asp	Glu	Thr	Glu	Ile	Thr	Asn	Pro	Val	Pro	Lys	Lys	Asn	
1400						1405					1410				
Val	Thr	Val	Lys	Lys	Thr	Ala	Ala	Lys	Ser	Gln	Ser	Ser	Thr	Ser	
1415						1420					1425				
Thr	Thr	Gly	Ala	Lys	Lys	Arg	Ala	Ala	Pro	Lys	Gly	Thr	Lys	Arg	
1430						1435					1440				

Asp	Pro	Ala	Leu	Asn	Ser	Gly	Val	Ser	Gln	Lys	Pro	Asp	Pro	Ala
1445						1450					1455			
Lys	Thr	Lys	Asn	Arg	Arg	Lys	Arg	Lys	Pro	Ser	Thr	Ser	Asp	Asp
1460						1465					1470			
Ser	Asp	Ser	Asn	Phe	Glu	Lys	Ile	Val	Ser	Lys	Ala	Val	Thr	Ser
1475						1480					1485			
Lys	Lys	Ser	Lys	Gly	Glu	Ser	Asp	Asp	Phe	His	Met	Asp	Phe	Asp
1490						1495					1500			
Ser	Ala	Val	Ala	Pro	Arg	Ala	Lys	Ser	Val	Arg	Ala	Lys	Lys	Pro
1505						1510					1515			
Ile	Lys	Tyr	Leu	Glu	Glu	Ser	Asp	Glu	Asp	Asp	Leu	Phe		
1520						1525					1530			

<210> 47

<211> 258

<212> PRT

<213> Homo sapiens

<400> 47

Met	Leu	Pro	Leu	Cys	Leu	Val	Ala	Ala	Leu	Leu	Leu	Ala	Ala	Gly	Pro
1			5						10					15	
Gly	Pro	Ser	Leu	Gly	Asp	Glu	Ala	Ile	His	Cys	Pro	Pro	Cys	Ser	Glu
			20					25					30		
Glu	Lys	Leu	Ala	Arg	Cys	Arg	Pro	Val	Gly	Cys	Glu	Glu	Leu	Val	
		35					40				45				
Arg	Glu	Pro	Gly	Cys	Gly	Cys	Cys	Ala	Thr	Cys	Ala	Leu	Gly	Leu	Gly
	50					55					60				
Met	Pro	Cys	Gly	Val	Tyr	Thr	Pro	Arg	Cys	Gly	Ser	Gly	Leu	Arg	Cys
65					70					75				80	
Tyr	Pro	Pro	Arg	Gly	Val	Glu	Lys	Pro	Leu	His	Thr	Leu	Met	His	Gly
			85						90					95	
Gln	Gly	Val	Cys	Met	Glu	Leu	Ala	Glu	Ile	Glu	Ala	Ile	Gln	Glu	Ser
			100					105					110		
Leu	Gln	Pro	Ser	Asp	Lys	Asp	Glu	Gly	Asp	His	Pro	Asn	Asn	Ser	Phe
		115					120					125			
Ser	Pro	Cys	Ser	Ala	His	Asp	Arg	Arg	Cys	Leu	Gln	Lys	His	Phe	Ala
	130					135					140				
Lys	Ile	Arg	Asp	Arg	Ser	Thr	Ser	Gly	Gly	Lys	Met	Lys	Val	Asn	Gly
145					150					155				160	
Ala	Pro	Arg	Glu	Asp	Ala	Arg	Pro	Val	Pro	Gln	Gly	Ser	Cys	Gln	Ser
			165						170					175	
Glu	Leu	His	Arg	Ala	Leu	Glu	Arg	Leu	Ala	Ala	Ser	Gln	Ser	Arg	Thr
			180					185					190		
His	Glu	Asp	Leu	Tyr	Ile	Ile	Pro	Ile	Pro	Asn	Cys	Asp	Arg	Asn	Gly
	195						200					205			
Asn	Phe	His	Pro	Lys	Gln	Cys	His	Pro	Ala	Leu	Asp	Gly	Gln	Arg	Gly
	210					215					220				
Lys	Cys	Trp	Cys	Val	Asp	Arg	Lys	Thr	Gly	Val	Lys	Leu	Pro	Gly	Gly
225					230					235				240	
Leu	Glu	Pro	Lys	Gly	Glu	Leu	Asp	Cys	His	Gln	Leu	Ala	Asp	Ser	Phe
				245					250					255	

Arg Glu

<210> 48

<211> 378

<212> PRT

<213> Homo sapiens

<400> 48
 Met Asp Leu Gly Lys Pro Met Lys Ser Val Leu Val Val Ala Leu Leu
 1 5 10 15
 Val Ile Phe Gln Val Cys Leu Cys Gln Asp Glu Val Thr Asp Asp Tyr
 20 25 30
 Ile Gly Asp Asn Thr Thr Val Asp Tyr Thr Leu Phe Glu Ser Leu Cys
 35 40 45
 Ser Lys Lys Asp Val Arg Asn Phe Lys Ala Trp Phe Leu Pro Ile Met
 50 55 60
 Tyr Ser Ile Ile Cys Phe Val Gly Leu Leu Gly Asn Gly Leu Val Val
 65 70 75 80
 Leu Thr Tyr Ile Tyr Phe Lys Arg Leu Lys Thr Met Thr Asp Thr Tyr
 85 90 95
 Leu Leu Asn Leu Ala Val Ala Asp Ile Leu Phe Leu Leu Thr Leu Pro
 100 105 110
 Phe Trp Ala Tyr Ser Ala Ala Lys Ser Trp Val Phe Gly Val His Phe
 115 120 125
 Cys Lys Leu Ile Phe Ala Ile Tyr Lys Met Ser Phe Phe Ser Gly Met
 130 135 140
 Leu Leu Leu Leu Cys Ile Ser Ile Asp Arg Tyr Val Ala Ile Val Gln
 145 150 155 160
 Ala Val Ser Ala His Arg His Arg Ala Arg Val Leu Leu Ile Ser Lys
 165 170 175
 Leu Ser Cys Val Gly Ile Trp Ile Leu Ala Thr Val Leu Ser Ile Pro
 180 185 190
 Glu Leu Leu Tyr Ser Asp Leu Gln Arg Ser Ser Ser Glu Gln Ala Met
 195 200 205
 Arg Cys Ser Leu Ile Thr Glu His Val Glu Ala Phe Ile Thr Ile Gln
 210 215 220
 Val Ala Gln Met Val Ile Gly Phe Leu Val Pro Leu Leu Ala Met Ser
 225 230 235 240
 Phe Cys Tyr Leu Val Ile Ile Arg Thr Leu Leu Gln Ala Arg Asn Phe
 245 250 255
 Glu Arg Asn Lys Ala Ile Lys Val Ile Ile Ala Val Val Val Val Phe
 260 265 270
 Ile Val Phe Gln Leu Pro Tyr Asn Gly Val Val Leu Ala Gln Thr Val
 275 280 285
 Ala Asn Phe Asn Ile Thr Ser Ser Thr Cys Glu Leu Ser Lys Gln Leu
 290 295 300
 Asn Ile Ala Tyr Asp Val Thr Tyr Ser Leu Ala Cys Val Arg Cys Cys
 305 310 315 320
 Val Asn Pro Phe Leu Tyr Ala Phe Ile Gly Val Lys Phe Arg Asn Asp
 325 330 335
 Leu Phe Lys Leu Phe Lys Asp Leu Gly Cys Leu Ser Gln Glu Gln Leu
 340 345 350
 Arg Gln Trp Ser Ser Cys Arg His Ile Arg Arg Ser Ser Met Ser Val
 355 360 365
 Glu Ala Glu Thr Thr Thr Thr Phe Ser Pro
 370 375

<210> 49

<211> 411

<212> PRT

<213> Homo sapiens

<400> 49
 Met Ser Lys Arg Pro Ser Tyr Ala Pro Pro Pro Thr Pro Ala Pro Ala
 1 5 10 15
 Thr Gln Met Pro Ser Thr Pro Gly Phe Val Gly Tyr Asn Pro Tyr Ser
 20 25 30

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His	Leu	Ala	Tyr	Asn	Asn	Tyr	Arg	Leu	Gly	Gly	Asn	Pro	Ser	Thr	Asn	
	35						40				45					
Ser	Arg	Val	Thr	Ala	Ser	Ser	Gly	Ile	Thr	Ile	Pro	Lys	Pro	Pro	Lys	
	50					55					60					
Pro	Pro	Asp	Lys	Pro	Leu	Met	Pro	Tyr	Met	Arg	Tyr	Ser	Arg	Lys	Val	
65					70					75					80	
Trp	Asp	Gln	Val	Lys	Ala	Ser	Asn	Pro	Asp	Leu	Lys	Leu	Trp	Glu	Ile	
				85					90					95		
Gly	Lys	Ile	Ile	Gly	Gly	Met	Trp	Arg	Asp	Leu	Thr	Asp	Glu	Glu	Lys	
			100					105					110			
Gln	Glu	Tyr	Leu	Asn	Glu	Tyr	Glu	Ala	Glu	Lys	Ile	Glu	Tyr	Asn	Glu	
		115					120					125				
Ser	Met	Lys	Ala	Tyr	His	Asn	Ser	Pro	Ala	Tyr	Leu	Ala	Tyr	Ile	Asn	
	130					135					140					
Ala	Lys	Ser	Arg	Ala	Glu	Ala	Ala	Leu	Glu	Glu	Glu	Ser	Arg	Gln	Arg	
145					150				155						160	
Gln	Ser	Arg	Met	Glu	Lys	Gly	Glu	Pro	Tyr	Met	Ser	Ile	Gln	Pro	Ala	
			165					170					175			
Glu	Asp	Pro	Asp	Asp	Tyr	Asp	Asp	Gly	Phe	Ser	Met	Lys	His	Thr	Ala	
			180					185					190			
Thr	Ala	Arg	Phe	Gln	Arg	Asn	His	Arg	Leu	Ile	Ser	Glu	Ile	Leu	Ser	
	195					200						205				
Glu	Ser	Val	Val	Pro	Asp	Val	Arg	Ser	Val	Val	Thr	Thr	Ala	Arg	Met	
	210				215						220					
Gln	Val	Leu	Lys	Arg	Gln	Val	Gln	Ser	Leu	Met	Val	His	Gln	Arg	Lys	
225					230				235						240	
Leu	Glu	Ala	Glu	Leu	Gln	Ile	Glu	Glu	Arg	His	Gln	Glu	Lys	Lys		
			245				250					255				
Arg	Lys	Phe	Leu	Glu	Ser	Thr	Asp	Ser	Phe	Asn	Asn	Glu	Leu	Lys	Arg	
		260					265					270				
Leu	Cys	Gly	Leu	Lys	Val	Glu	Val	Asp	Met	Glu	Lys	Ile	Ala	Ala	Glu	
	275					280					285					
Ile	Ala	Gln	Ala	Glu	Glu	Gln	Ala	Arg	Lys	Arg	Gln	Glu	Glu	Arg	Glu	
	290				295						300					
Lys	Glu	Ala	Ala	Glu	Gln	Ala	Glu	Arg	Ser	Gln	Ser	Ser	Ile	Val	Pro	
305					310				315						320	
Glu	Glu	Glu	Gln	Ala	Asn	Lys	Gly	Glu	Glu	Lys	Lys	Asp	Asp	Glu		
			325				330					335				
Asn	Ile	Pro	Met	Glu	Thr	Glu	Glu	Thr	His	Leu	Glu	Glu	Thr	Thr	Glu	
			340				345					350				
Ser	Gln	Gln	Asn	Gly	Glu	Glu	Gly	Thr	Ser	Thr	Pro	Glu	Asp	Lys	Glu	
	355					360					365					
Ser	Gly	Gln	Glu	Gly	Val	Asp	Ser	Met	Ala	Glu	Glu	Gly	Thr	Ser	Asp	
	370				375						380					
Ser	Asn	Thr	Gly	Ser	Glu	Ser	Asn	Ser	Ala	Thr	Val	Glu	Glu	Pro	Pro	
385					390				395						400	
Thr	Asp	Pro	Ile	Pro	Glu	Asp	Glu	Lys	Lys	Glu						
				405					410							

<210> 50

<211> 593

<212> PRT

<213> Homo sapiens

<400> 50

Met	Ser	Val	Arg	Tyr	Ser	Ser	Ser	Lys	His	Tyr	Ser	Ser	Ser	Arg	Ser	
1				5					10					15		
Gly	Gly	Gly	Gly	Gly	Gly	Gly	Gly	Cys	Gly	Gly	Gly	Gly	Gly	Val	Ser	
			20					25					30			
Ser	Leu	Arg	Ile	Ser	Ser	Ser	Lys	Gly	Ser	Leu	Gly	Gly	Gly	Phe	Ser	
	35						40					45				

Ser Gly Gly Phe Ser Gly Gly Ser Phe Ser Arg Gly Ser Ser Gly Gly
 50 55 60
 Gly Cys Phe Gly Gly Ser Ser Gly Gly Tyr Gly Gly Leu Gly Gly Phe
 65 70 75 80
 Gly Gly Gly Ser Phe His Gly Ser Tyr Gly Ser Ser Ser Phe Gly Gly
 85 90 95
 Ser Tyr Gly Gly Ser Phe Gly Gly Gly Asn Phe Gly Gly Gly Ser Phe
 100 105 110
 Gly Gly Gly Ser Phe Gly Gly Gly Phe Gly Gly Gly Phe Gly
 115 120 125
 Gly Gly Phe Gly Gly Gly Phe Gly Gly Asp Gly Gly Leu Leu Ser Gly
 130 135 140
 Asn Glu Lys Val Thr Met Gln Asn Leu Asn Asp Arg Leu Ala Ser Tyr
 145 150 155 160
 Leu Asp Lys Val Arg Ala Leu Glu Glu Ser Asn Tyr Glu Leu Glu Gly
 165 170 175
 Lys Ile Lys Glu Trp Tyr Glu Lys His Gly Asn Ser His Gln Gly Glu
 180 185 190
 Pro Arg Asp Tyr Ser Lys Tyr Tyr Lys Thr Ile Asp Asp Leu Lys Asn
 195 200 205
 Gln Ile Leu Asn Leu Thr Thr Asp Asn Ala Asn Ile Leu Leu Gln Ile
 210 215 220
 Asp Asn Ala Arg Leu Ala Ala Asp Asp Phe Arg Leu Lys Tyr Glu Asn
 225 230 235 240
 Glu Val Ala Leu Arg Gln Ser Val Glu Ala Asp Ile Asn Gly Leu Arg
 245 250 255
 Arg Val Leu Asp Glu Leu Thr Leu Thr Lys Ala Asp Leu Glu Met Gln
 260 265 270
 Ile Glu Ser Leu Thr Glu Glu Leu Ala Tyr Leu Lys Lys Asn His Glu
 275 280 285
 Glu Glu Met Lys Asp Leu Arg Asn Val Ser Thr Gly Asp Val Asn Val
 290 295 300
 Glu Met Asn Ala Ala Pro Gly Val Asp Leu Thr Gln Leu Leu Asn Asn
 305 310 315 320
 Met Arg Ser Gln Tyr Glu Gln Leu Ala Glu Gln Asn Arg Lys Asp Ala
 325 330 335
 Glu Ala Trp Phe Asn Glu Lys Ser Lys Glu Leu Thr Thr Glu Ile Asp
 340 345 350
 Asn Asn Ile Glu Gln Ile Ser Ser Tyr Lys Ser Glu Ile Thr Glu Leu
 355 360 365
 Arg Arg Asn Val Gln Ala Leu Glu Ile Glu Leu Gln Ser Gln Leu Ala
 370 375 380
 Leu Lys Gln Ser Leu Glu Ala Ser Leu Ala Glu Thr Glu Gly Arg Tyr
 385 390 395 400
 Cys Val Gln Leu Ser Gln Ile His Ala Gln Ile Ser Ala Leu Glu Glu
 405 410 415
 Gln Leu Gln Gln Ile Arg Ala Glu Thr Glu Cys Gln Asn Thr Glu Tyr
 420 425 430
 Gln Gln Leu Asp Ile Lys Ile Arg Leu Glu Asn Glu Ile Gln Thr
 435 440 445
 Tyr Arg Ser Leu Leu Glu Gly Glu Gly Ser Ser Gly Gly Gly Arg
 450 455 460
 Gly Gly Gly Ser Phe Gly Gly Gly Tyr Gly Gly Gly Ser Ser Gly Gly
 465 470 475 480
 Gly Ser Ser Gly Gly Tyr Gly Gly Gly His Gly Gly Ser Ser Gly
 485 490 495
 Gly Gly Tyr Gly Gly Gly Ser Ser Gly Gly Gly Ser Ser Gly Gly
 500 505 510
 Tyr Gly Gly Gly Ser Ser Ser Gly Gly His Gly Gly Gly Ser Ser Ser
 515 520 525
 Gly Gly His Gly Gly Ser Ser Ser Gly Gly Tyr Gly Gly Gly Ser Ser
 530 535 540
 Gly Gly Gly Gly Gly Gly Tyr Gly Gly Gly Ser Ser Gly Gly Gly Ser
 545 550 555 560
 Ser Ser Gly Gly Gly Tyr Gly Gly Gly Ser Ser Ser Gly Gly His Lys
 565 570 575

Ser Ser Ser Ser Gly Ser Val Gly Glu Ser Ser Ser Lys Gly Pro Arg
 580 585 590
 Tyr

<210> 51

<211> 494

<212> PRT

<213> Homo sapiens

<400> 51

Met	Asp	Leu	Ser	Asn	Asn	Thr	Met	Ser	Leu	Ser	Val	Arg	Thr	Pro	Gly
1				5					10					15	
Leu	Ser	Arg	Arg	Leu	Ser	Ser	Gln	Ser	Val	Ile	Gly	Arg	Pro	Arg	Gly
			20					25					30		
Met	Ser	Ala	Ser	Ser	Val	Gly	Ser	Gly	Tyr	Gly	Gly	Ser	Ala	Phe	Gly
		35					40					45			
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Glu	Leu	Thr	Leu	Thr	Arg	Thr	Asp	Leu	Glu	Met	Gln	Ile	Glu	Ser	Leu
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Val Arg Ala Asp Ala Glu Arg Gln Asn Val Asp His Gln Arg Leu Leu
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 Asn Val Lys Ala Arg Leu Glu Leu Glu Ile Glu Thr Tyr Arg Arg Leu
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 Leu Asp Gly Glu Ala Gln Gly Asp Gly Leu Glu Glu Ser Leu Phe Val
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 Thr Asp Ser Lys Ser Gln Ala Gln Ser Thr Asp Ser Ser Lys Asp Pro
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 Thr Lys Thr Arg Lys Ile Lys Thr Val Val Gln Glu Met Val Asn Gly
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<210> 52

<211> 361

<212> PRT

<213> Homo sapiens

<400> 52

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 Ser Gln Gln Gln Glu Pro Leu Leu Cys Pro Ser Tyr Gln Ser Tyr Phe
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 Lys Thr Ile Glu Glu Leu Gln Gln Lys Ile Leu Cys Ser Lys Ser Glu
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 Asn Ala Arg Leu Val Val Gln Ile Asp Asn Ala Lys Leu Ala Ala Asp
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 Asp Phe Arg Thr Lys Tyr Gln Thr Glu Gln Ser Leu Arg Gln Leu Val
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 Glu Ser Asp Ile Asn Ser Leu Arg Arg Ile Leu Asp Glu Leu Thr Leu
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 Cys Arg Ser Asp Leu Glu Ala Gln Met Glu Ser Leu Lys Glu Glu Leu
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 Leu Ser Leu Lys Gln Asn His Glu Gln Glu Val Asn Thr Leu Arg Cys
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 Gln Leu Gly Asp Arg Leu Asn Val Glu Val Asp Ala Ala Pro Ala Val
 165 170 175
 Asp Leu Asn Gln Val Leu Asn Glu Thr Arg Asn Gln Tyr Glu Ala Leu
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 Val Glu Thr Asn Arg Arg Glu Val Glu Gln Trp Phe Ala Thr Gln Thr
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 Ile Glu Leu Gln Ala Gln His Asn Leu Arg Tyr Ser Leu Glu Asn Thr
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 Arg Leu Glu Cys Glu Ile Asn Thr Tyr Arg Ser Leu Leu Glu Ser Glu
 305 310 315 320
 Asp Cys Lys Leu Pro Ser Asn Pro Cys Ala Thr Thr Asn Ala Cys Glu
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<210> 53

<211> 3282

<212> DNA

<213> Homo sapiens

<400> 53

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<210> 54

<211> 2227

<212> DNA

<213> Homo sapiens

<400> 54

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<210> 55

<211> 4283

<212> DNA

<213> Homo sapiens

<400> 55

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<210> 59

<211> 784

<212> DNA

<213> Homo sapiens

<400> 59

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<210> 60

<211> 3033

<212> DNA

<213> Homo sapiens

<400> 60

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<210> 61

<211> 1174

<212> DNA

<213> Homo sapiens

<400> 61

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<211> 3167

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<213> Homo sapiens

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<210> 63

<211> 2733

<212> DNA

<213> Homo sapiens

<220>

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<223> n=a, c, g or t

<220>

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<222> (2724) .. (2724)

<223> n=a, c, g or t

<400> 63

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<211> 2546

<212> DNA

<213> Homo sapiens

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<211> 1841

<212> DNA

<213> Homo sapiens

<400> 69

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<211> 748

<212> DNA

<213> Homo sapiens

<400> 70
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<211> 795

<212> DNA

<213> Homo sapiens

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<210> 72

<211> 2356

<212> DNA

<213> Homo sapiens

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<210> 73

<211> 1646

<212> DNA

<213> Homo sapiens

<400> 73

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<211> 3340

<212> DNA

<213> Homo sapiens

<400> 74

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<211> 4005

<212> DNA

<213> Homo sapiens

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agccttggct	cagtgtccct	gtgtacaaga	cccagtgaat	tccaggctcc	cagaaacccc	1140
accctaacca	tgggccaacc	cagaacaccc	cactctccac	cactggccaa	agaacatgcc	1200
agcatctgcc	ccccatccat	caccaactcc	atggtggaca	taccatttgt	gctgatcaac	1260
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caacctggag	ctgcttctcc	cagcaacccc	tgtccagcca	ccaggagcaa	cagccagacc	1380
ctgtcagatg	ccccctttac	cacatgccc	gagggtccc	ccaggagcat	gcagcccacc	1440
atgaagtctg	tgatggacac	atctaaatac	tggtttaagc	caaacatcac	ccgagagcaa	1500
gcaatcgagc	tgttgaggaa	ggaggagcca	ggggcttttg	tcataaggga	cagctcttca	1560
taccagggtc	ccttcggcct	ggccctgaag	gtgcaggagg	ttcccgcgtc	tgctcagaat	1620
cgaccagggtg	aggacagcaa	tgacctcatc	cgacacttcc	tcacgcagtc	gtctgccaaa	1680
ggagtgcac	tcaaaggagc	agatgaggag	ccctactttg	ggagcctctc	tgccttcgtg	1740
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ctgggagggtg	cagatggggc	ctcggactct	acagacagcc	cagcctcctg	ccagaagaaa	1860
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ctggccctgc	agaaagccat	ctccaccacc	tttgagaggg	acatcctccc	cagcccccacc	1980
gtggctcact	tcgaagtcac	agagcagggc	atcactctga	ctgatgtcca	gaggaagggtg	2040
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cggaagtggc	agaagtactg	caaaccctcc	tggatctttg	ggtttgtggc	caagagccag	2160
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acaacagctc	gagcaagaga	cctgcagccc	ctgtttcgtg	gcagacagca	gggtgcctggc	2700
ggtagccac	ggggctcctg	gcttgcagct	gggtgatggc	aagaactgac	tacaaaacag	2760
gaatggatag	actctatttc	cttccatata	tgttcctctg	ttccttttcc	cactttcttg	2820
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gcagctcagc	agggggaact	tgtccccatg	gtcagaggag	acccagctgt	cctgcacccc	2940
cttgagatg	agtatcaccc	catcttttct	ttccacttgg	ttttttattt	tatttttttt	3000
gagacagagt	ctcactgtca	cccaggctga	actgcagtgg	tgtgatctag	gctcactgca	3060
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caggcatgtg	caactcaccc	agctaatttt	gtatttttag	tagagacagg	gtttccacat	3180
gttggccagg	ctggtcctga	actcctgacc	gcaggtaatc	cacctgcttc	ggcctcccaa	3240
agtgtctggga	ttacaggcgc	aagccaccca	gcccagcttc	tttccattcc	ttgataggcg	3300

agtattccaa	agctggtatc	gtagctgccc	taatgttgca	tattaggcgg	cgggggcaga	3360
gataagggcc	atctctctgt	gattctgcct	cagctcctgt	cttgctgagc	cctcccccaa	3420
cccacgctcc	aacacacaca	cacacacaca	cacacacaca	cacacacaca	cacacacaca	3480
cacgccccctc	tactgctatg	tggtttcaac	cagcctcaca	gccacacggg	ggaagcagag	3540
agtcaagaat	gcaaagaggc	cgcttcctta	agaggcttgg	aggagctggg	ctctatccca	3600
caccaccccc	caccaccccc	ccaccagccc	tccagaagct	ggaaccattt	ctcccgcagg	3660
cctgagttcc	taaggaaacc	accctaccgg	ggtggaaggg	agggtcaggg	aagaaaccca	3720
ctcttgctct	acgaggagca	agtgcctgcc	ccctcccagc	agccagccct	gccaaagttg	3780
cattatcttt	ggccaaggct	gggcctgacg	gttatgattt	cagccctggg	cctgcaggag	3840
aggctgagat	cagccccacc	agccagtggg	cgagcactgc	cccgccgcca	aagtctgcag	3900
aatgtgagat	gaggttctca	aggtcacagg	ccccagtccc	agcctggggg	ctggcagagg	3960
ccccatata	ctctgctaca	gctcctatca	tgaaaaataa	aatgt		4005

<210> 76

<211> 1093

<212> PRT

<213> Homo sapiens

<400> 76

Met	Lys	Glu	Met	Val	Gly	Gly	Cys	Cys	Val	Cys	Ser	Asp	Glu	Arg	Gly
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Trp	Ala	Glu	Asn	Pro	Leu	Val	Tyr	Cys	Asp	Gly	His	Ala	Cys	Ser	Val
			20					25					30		
Ala	Val	His	Gln	Ala	Cys	Tyr	Gly	Ile	Val	Gln	Val	Pro	Thr	Gly	Pro
		35					40				45				
Trp	Phe	Cys	Arg	Lys	Cys	Glu	Ser	Gln	Glu	Arg	Ala	Ala	Arg	Val	Arg
	50				55						60				
Cys	Glu	Leu	Cys	Pro	His	Lys	Asp	Gly	Ala	Leu	Lys	Arg	Thr	Asp	Asn
65					70					75					80
Gly	Gly	Trp	Ala	His	Val	Val	Cys	Ala	Leu	Tyr	Ile	Pro	Glu	Val	Gln
			85						90					95	
Phe	Ala	Asn	Val	Leu	Thr	Met	Glu	Pro	Ile	Val	Leu	Gln	Tyr	Val	Pro
			100					105					110		
His	Asp	Arg	Phe	Asn	Lys	Thr	Cys	Tyr	Ile	Cys	Glu	Glu	Thr	Gly	Arg
		115					120					125			
Glu	Ser	Lys	Ala	Ala	Ser	Gly	Ala	Cys	Met	Thr	Cys	Asn	Arg	His	Gly
	130					135					140				
Cys	Arg	Gln	Ala	Phe	His	Val	Thr	Cys	Ala	Gln	Met	Ala	Gly	Leu	Leu
145					150					155					160
Cys	Glu	Glu	Glu	Val	Leu	Glu	Val	Asp	Asn	Val	Lys	Tyr	Cys	Gly	Tyr
			165						170					175	
Cys	Lys	Tyr	His	Phe	Ser	Lys	Met	Lys	Thr	Ser	Arg	His	Ser	Ser	Gly
			180					185					190		
Gly	Gly	Gly	Gly	Gly	Ala	Gly	Gly	Gly	Gly	Gly	Ser	Met	Gly	Gly	Gly
	195					200						205			
Gly	Ser	Gly	Phe	Ile	Ser	Gly	Arg	Arg	Ser	Arg	Ser	Ala	Ser	Pro	Ser
	210					215						220			
Thr	Gln	Gln	Glu	Lys	His	Pro	Thr	His	His	Glu	Arg	Gly	Gln	Lys	Lys
225					230					235					240
Ser	Arg	Lys	Asp	Lys	Glu	Arg	Leu	Lys	Gln	Lys	His	Lys	Lys	Arg	Pro
				245					250					255	
Glu	Ser	Pro	Pro	Ser	Ile	Leu	Thr	Pro	Pro	Val	Val	Pro	Thr	Ala	Asp
		260						265					270		
Lys	Val	Ser	Ser	Ser	Ala	Ser	Ser	Ser	Ser	His	His	Glu	Ala	Ser	Thr
	275						280					285			
Gln	Glu	Thr	Ser	Glu	Ser	Ser	Arg	Glu	Ser	Lys	Gly	Lys	Lys	Ser	Ser
	290					295					300				
Ser	His	Ser	Leu	Ser	His	Lys	Gly	Lys	Lys	Leu	Ser	Ser	Gly	Lys	Gly
305					310					315					320
Val	Ser	Ser	Phe	Thr	Ser	Ala	Ser	Ser	Ser	Ser	Ser	Ser	Ser	Ser	Ser
				325					330						335

Ser Ser Gly Gly Pro Phe Gln Pro Ala Val Ser Ser Leu Gln Ser Ser
 340 345 350
 Pro Asp Phe Ser Ala Phe Pro Lys Leu Glu Gln Pro Glu Glu Asp Lys
 355 360 365
 Tyr Ser Lys Pro Thr Ala Pro Ala Pro Ser Ala Pro Pro Ser Pro Ser
 370 375 380
 Ala Pro Glu Pro Pro Lys Ala Asp Leu Phe Glu Gln Lys Val Val Phe
 385 390 395 400
 Ser Gly Phe Gly Pro Ile Met Arg Phe Ser Thr Thr Thr Ser Ser Ser
 405 410 415
 Gly Arg Ala Arg Ala Pro Ser Pro Gly Asp Tyr Lys Ser Pro His Val
 420 425 430
 Thr Gly Ser Gly Ala Ser Ala Gly Thr His Lys Arg Met Pro Ala Leu
 435 440 445
 Ser Ala Thr Pro Val Pro Ala Asp Glu Thr Pro Glu Thr Gly Leu Lys
 450 455 460
 Glu Lys Lys His Lys Ala Ser Lys Arg Ser Arg His Gly Pro Gly Arg
 465 470 475 480
 Pro Lys Gly Ser Arg Asn Lys Glu Gly Thr Gly Gly Pro Ala Ala Pro
 485 490 495
 Ser Leu Pro Ser Ala Gln Leu Ala Gly Phe Thr Ala Thr Ala Ala Ser
 500 505 510
 Pro Phe Ser Gly Gly Ser Leu Val Ser Ser Gly Leu Gly Gly Leu Ser
 515 520 525
 Ser Arg Thr Phe Gly Pro Ser Gly Ser Leu Pro Ser Leu Ser Leu Glu
 530 535 540
 Ser Pro Leu Leu Gly Ala Gly Ile Tyr Thr Ser Asn Lys Asp Pro Ile
 545 550 555 560
 Ser His Ser Gly Gly Met Leu Arg Ala Val Cys Ser Thr Pro Leu Ser
 565 570 575
 Ser Ser Leu Leu Gly Pro Pro Gly Thr Ser Ala Leu Pro Arg Leu Ser
 580 585 590
 Arg Ser Pro Phe Thr Ser Thr Leu Pro Ser Ser Ser Ala Ser Ile Ser
 595 600 605
 Thr Thr Gln Val Phe Ser Leu Ala Gly Ser Thr Phe Ser Leu Pro Ser
 610 615 620
 Thr His Ile Phe Gly Thr Pro Met Gly Ala Val Asn Pro Leu Leu Ser
 625 630 635 640
 Gln Ala Glu Ser Ser His Thr Glu Pro Asp Leu Glu Asp Cys Ser Phe
 645 650 655
 Arg Cys Arg Gly Thr Ser Pro Gln Glu Ser Leu Ser Ser Met Ser Pro
 660 665 670
 Ile Ser Ser Leu Pro Ala Leu Phe Asp Gln Thr Ala Ser Ala Pro Cys
 675 680 685
 Gly Gly Gly Gln Leu Asp Pro Ala Ala Pro Gly Thr Thr Asn Met Glu
 690 695 700
 Gln Leu Leu Glu Lys Gln Gly Asp Gly Glu Ala Gly Val Asn Ile Val
 705 710 715 720
 Glu Met Leu Lys Ala Leu His Ala Leu Gln Lys Glu Asn Gln Arg Leu
 725 730 735
 Gln Glu Gln Ile Leu Ser Leu Thr Ala Lys Lys Glu Arg Leu Gln Ile
 740 745 750
 Leu Asn Val Gln Leu Ser Val Pro Phe Pro Ala Leu Pro Ala Ala Leu
 755 760 765
 Pro Ala Ala Asn Gly Pro Val Pro Gly Pro Tyr Gly Leu Pro Pro Gln
 770 775 780
 Ala Gly Ser Ser Asp Ser Leu Ser Thr Ser Lys Ser Pro Pro Gly Lys
 785 790 795 800
 Ser Ser Leu Gly Leu Asp Asn Ser Leu Ser Thr Ser Ser Glu Asp Pro
 805 810 815
 His Ser Gly Cys Pro Ser Arg Ser Ser Ser Ser Leu Ser Phe His Ser
 820 825 830
 Thr Pro Pro Pro Leu Pro Leu Leu Gln Gln Ser Pro Ala Thr Leu Pro
 835 840 845
 Leu Ala Leu Pro Gly Ala Pro Ala Pro Leu Pro Pro Gln Pro Gln Asn
 850 855 860

Gly Leu Gly Arg Ala Pro Gly Ala Ala Gly Leu Gly Ala Met Pro Met
 865 870 875 880
 Ala Glu Gly Leu Leu Gly Gly Leu Ala Gly Ser Gly Gly Leu Pro Leu
 885 890 895
 Asn Gly Leu Leu Gly Gly Leu Asn Gly Ala Ala Ala Pro Asn Pro Ala
 900 905 910
 Ser Leu Ser Gln Ala Gly Gly Ala Pro Thr Leu Gln Leu Pro Gly Cys
 915 920 925
 Leu Asn Ser Leu Thr Glu Gln Gln Arg His Leu Leu Gln Gln Gln Glu
 930 935 940
 Gln Gln Leu Gln Gln Leu Gln Gln Leu Leu Ala Ser Pro Gln Leu Thr
 945 950 955 960
 Pro Glu His Gln Thr Val Val Tyr Gln Met Ile Gln Gln Ile Gln Gln
 965 970 975
 Lys Arg Glu Leu Gln Arg Leu Gln Met Ala Gly Gly Ser Gln Leu Pro
 980 985 990
 Met Ala Ser Leu Leu Ala Gly Ser Ser Thr Pro Leu Leu Ser Ala Gly
 995 1000 1005
 Thr Pro Gly Leu Leu Pro Thr Ala Ser Ala Pro Pro Leu Leu Pro
 1010 1015 1020
 Ala Gly Ala Leu Val Ala Pro Ser Leu Gly Asn Asn Thr Ser Leu
 1025 1030 1035
 Met Ala Ala Ala Ala Ala Ala Ala Ala Val Ala Ala Ala Gly Gly
 1040 1045 1050
 Pro Pro Val Leu Thr Ala Gln Thr Asn Pro Phe Leu Ser Leu Ser
 1055 1060 1065
 Gly Ala Glu Gly Ser Gly Gly Gly Pro Lys Gly Gly Thr Ala Asp
 1070 1075 1080
 Lys Gly Ala Ser Ala Asn Gln Glu Lys Gly
 1085 1090

<210> 77

<211> 344

<212> PRT

<213> Homo sapiens

<400> 77

Met His Arg Thr Thr Arg Ile Lys Ile Thr Glu Leu Asn Pro His Leu
 1 5 10 15
 Met Cys Ala Leu Cys Gly Gly Tyr Phe Ile Asp Ala Thr Thr Ile Val
 20 25 30
 Glu Cys Leu His Ser Phe Cys Lys Thr Cys Ile Val Arg Tyr Leu Glu
 35 40 45
 Thr Asn Lys Tyr Cys Pro Met Cys Asp Val Gln Val His Lys Thr Arg
 50 55 60
 Pro Leu Leu Ser Ile Arg Ser Asp Lys Thr Leu Gln Asp Ile Val Tyr
 65 70 75 80
 Lys Leu Val Pro Gly Leu Phe Lys Asp Glu Met Lys Arg Arg Arg Asp
 85 90 95
 Phe Tyr Ala Ala Tyr Pro Leu Thr Glu Val Pro Asn Gly Ser Asn Glu
 100 105 110
 Asp Arg Gly Glu Val Leu Glu Gln Glu Lys Gly Ala Leu Ser Asp Asp
 115 120 125
 Glu Ile Val Ser Leu Ser Ile Glu Phe Tyr Glu Gly Ala Arg Asp Arg
 130 135 140
 Asp Glu Lys Lys Gly Pro Leu Glu Asn Gly Asp Gly Asp Lys Glu Lys
 145 150 155 160
 Thr Gly Val Arg Phe Leu Arg Cys Pro Ala Ala Met Thr Val Met His
 165 170 175
 Leu Ala Lys Phe Leu Arg Asn Lys Met Asp Val Pro Ser Lys Tyr Lys
 180 185 190

Val Glu Val Leu Tyr Glu Asp Glu Pro Leu Lys Glu Tyr Tyr Thr Leu
 195 200 205
 Met Asp Ile Ala Tyr Ile Tyr Pro Trp Arg Arg Asn Gly Pro Leu Pro
 210 215 220
 Leu Lys Tyr Arg Val Gln Pro Ala Cys Lys Arg Leu Thr Leu Ala Thr
 225 230 235 240
 Val Pro Thr Pro Ser Glu Gly Thr Asn Thr Ser Gly Ala Ser Glu Cys
 245 250 255
 Glu Ser Val Ser Asp Lys Ala Pro Ser Pro Ala Thr Leu Pro Ala Thr
 260 265 270
 Ser Ser Ser Leu Pro Ser Pro Ala Thr Pro Ser His Gly Ser Pro Ser
 275 280 285
 Ser His Gly Pro Pro Ala Thr His Pro Thr Ser Pro Thr Pro Pro Ser
 290 295 300
 Thr Ala Ser Gly Ala Thr Thr Ala Ala Asn Gly Gly Ser Leu Asn Cys
 305 310 315 320
 Leu Gln Thr Pro Ser Ser Thr Ser Arg Gly Arg Lys Met Thr Val Asn
 325 330 335
 Gly Ala Pro Val Pro Pro Leu Thr
 340

<210> 78

<211> 416

<212> PRT

<213> Homo sapiens

<400> 78
 Met Ser Ser Asn Cys Thr Ser Thr Thr Ala Val Ala Val Ala Pro Leu
 1 5 10 15
 Ser Ala Ser Lys Thr Lys Thr Lys Lys Lys His Phe Val Cys Gln Lys
 20 25 30
 Val Lys Leu Phe Arg Ala Ser Glu Pro Ile Leu Ser Val Leu Met Trp
 35 40 45
 Gly Val Asn His Thr Ile Asn Glu Leu Ser Asn Val Pro Val Pro Val
 50 55 60
 Met Leu Met Pro Asp Asp Phe Lys Ala Tyr Ser Lys Ile Lys Val Asp
 65 70 75 80
 Asn His Leu Phe Asn Lys Glu Asn Leu Pro Ser Arg Phe Lys Phe Lys
 85 90 95
 Glu Tyr Cys Pro Met Val Phe Arg Asn Leu Arg Glu Arg Phe Gly Ile
 100 105 110
 Asp Asp Gln Asp Tyr Gln Asn Ser Val Thr Arg Ser Ala Pro Ile Asn
 115 120 125
 Ser Asp Ser Gln Gly Arg Cys Gly Thr Arg Phe Leu Thr Thr Tyr Asp
 130 135 140
 Arg Arg Phe Val Ile Lys Thr Val Ser Ser Glu Asp Val Ala Glu Met
 145 150 155 160
 His Asn Ile Leu Lys Lys Tyr His Gln Phe Ile Val Glu Cys His Gly
 165 170 175
 Asn Thr Leu Leu Pro Gln Phe Leu Gly Met Tyr Arg Leu Thr Val Asp
 180 185 190
 Gly Val Glu Thr Tyr Met Val Val Thr Arg Asn Val Phe Ser His Arg
 195 200 205
 Leu Thr Val His Arg Lys Tyr Asp Leu Lys Gly Ser Thr Val Ala Arg
 210 215 220
 Glu Ala Ser Asp Lys Glu Lys Ala Lys Asp Leu Pro Thr Phe Lys Asp
 225 230 235 240
 Asn Asp Phe Leu Asn Glu Gly Gln Lys Leu His Val Gly Glu Glu Ser
 245 250 255
 Lys Lys Asn Phe Leu Glu Lys Leu Lys Arg Asp Val Glu Phe Leu Ala
 260 265 270

Gln Leu Lys Ile Met Asp Tyr Ser Leu Leu Val Gly Ile His Asp Val
 275 280 285
 Asp Arg Ala Glu Gln Glu Glu Met Glu Val Glu Glu Arg Ala Glu Asp
 290 295 300
 Glu Glu Cys Glu Asn Asp Gly Val Gly Gly Asn Leu Leu Cys Ser Tyr
 305 310 315 320
 Gly Thr Pro Pro Asp Ser Pro Gly Asn Leu Leu Ser Phe Pro Arg Phe
 325 330 335
 Phe Gly Pro Gly Glu Phe Asp Pro Ser Val Asp Val Tyr Ala Met Lys
 340 345 350
 Ser His Glu Ser Ser Pro Lys Lys Glu Val Tyr Phe Met Ala Ile Ile
 355 360 365
 Asp Ile Leu Thr Pro Tyr Asp Thr Lys Lys Lys Ala Ala His Ala Ala
 370 375 380
 Lys Thr Val Lys His Gly Ala Gly Ala Glu Ile Ser Thr Val Asn Pro
 385 390 395 400
 Glu Gln Tyr Ser Lys Arg Phe Asn Glu Phe Met Ser Asn Ile Leu Thr
 405 410 415

<210> 79

<211> 500

<212> PRT

<213> Homo sapiens

<400> 79

Met Arg Gly Glu Leu Trp Leu Leu Val Leu Val Leu Arg Glu Ala Ala
 1 5 10 15
 Arg Ala Leu Ser Pro Gln Pro Gly Ala Gly His Asp Glu Gly Pro Gly
 20 25 30
 Ser Gly Trp Ala Ala Lys Gly Thr Val Arg Gly Trp Asn Arg Arg Ala
 35 40 45
 Arg Glu Ser Pro Gly His Val Ser Glu Pro Asp Arg Thr Gln Leu Ser
 50 55 60
 Gln Asp Leu Gly Gly Gly Thr Leu Ala Met Asp Thr Leu Pro Asp Asn
 65 70 75 80
 Arg Thr Arg Val Val Glu Asp Asn His Ser Tyr Tyr Val Ser Arg Leu
 85 90 95
 Tyr Gly Pro Ser Glu Pro His Ser Arg Glu Leu Trp Val Asp Val Ala
 100 105 110
 Glu Ala Asn Arg Ser Gln Val Lys Ile His Thr Ile Leu Ser Asn Thr
 115 120 125
 His Arg Gln Ala Ser Arg Val Val Leu Ser Phe Asp Phe Pro Phe Tyr
 130 135 140
 Gly His Pro Leu Arg Gln Ile Thr Ile Ala Thr Gly Gly Phe Ile Phe
 145 150 155 160
 Met Gly Asp Val Ile His Arg Met Leu Thr Ala Thr Gln Tyr Val Ala
 165 170 175
 Pro Leu Met Ala Asn Phe Asn Pro Gly Tyr Ser Asp Asn Ser Thr Val
 180 185 190
 Val Tyr Phe Asp Asn Gly Thr Val Phe Val Val Gln Trp Asp His Val
 195 200 205
 Tyr Leu Gln Gly Trp Glu Asp Lys Gly Ser Phe Thr Phe Gln Ala Ala
 210 215 220
 Leu His His Asp Gly Arg Ile Val Phe Ala Tyr Lys Glu Ile Pro Met
 225 230 235 240
 Ser Val Pro Glu Ile Ser Ser Ser Gln His Pro Val Lys Thr Gly Leu
 245 250 255
 Ser Asp Ala Phe Met Ile Leu Asn Pro Ser Pro Asp Val Pro Glu Ser
 260 265 270
 Arg Arg Arg Ser Ile Phe Glu Tyr His Arg Ile Glu Leu Asp Pro Ser
 275 280 285

Lys Val Thr Ser Met Ser Ala Val Glu Phe Thr Pro Leu Pro Thr Cys
 290 295 300
 Leu Gln His Arg Ser Cys Asp Ala Cys Met Ser Ser Asp Leu Thr Phe
 305 310 315 320
 Asn Cys Ser Trp Cys His Val Leu Gln Arg Cys Ser Ser Gly Phe Asp
 325 330 335
 Arg Tyr Arg Gln Glu Trp Met Asp Tyr Gly Cys Ala Gln Glu Ala Glu
 340 345 350
 Gly Arg Met Cys Glu Asp Phe Gln Asp Glu Asp His Asp Ser Ala Ser
 355 360 365
 Pro Asp Thr Ser Phe Ser Pro Tyr Asp Gly Asp Leu Thr Thr Thr Ser
 370 375 380
 Ser Ser Leu Phe Ile Asp Ser Leu Thr Thr Glu Asp Asp Thr Lys Leu
 385 390 395 400
 Asn Pro Tyr Ala Gly Asp Gly Leu Gln Asn Asn Leu Ser Pro Lys
 405 410 415
 Thr Lys Gly Thr Pro Val His Leu Gly Thr Ile Val Gly Ile Val Leu
 420 425 430
 Ala Val Leu Leu Val Ala Ala Ile Ile Leu Ala Gly Ile Tyr Ile Asn
 435 440 445
 Gly His Pro Thr Ser Asn Ala Ala Leu Phe Phe Ile Glu Arg Arg Pro
 450 455 460
 His His Trp Pro Ala Met Lys Phe Arg Ser His Pro Asp His Ser Thr
 465 470 475 480
 Tyr Ala Glu Val Glu Pro Ser Gly His Glu Lys Glu Gly Phe Met Glu
 485 490 495
 Ala Glu Gln Cys
 500

<210> 80

<211> 509

<212> PRT

<213> Homo sapiens

<400> 80

Met Glu Asp Ile Gln Thr Asn Ala Glu Leu Lys Ser Thr Gln Glu Gln
 1 5 10 15
 Ser Val Pro Ala Glu Ser Ala Ala Val Leu Asn Asp Tyr Ser Leu Thr
 20 25 30
 Lys Ser His Glu Met Glu Asn Val Asp Ser Gly Glu Gly Pro Ala Asn
 35 40 45
 Glu Asp Glu Asp Ile Gly Asp Asp Ser Met Lys Val Lys Asp Glu Tyr
 50 55 60
 Ser Glu Arg Asp Glu Asn Val Leu Lys Ser Glu Pro Met Gly Asn Ala
 65 70 75 80
 Glu Glu Pro Glu Ile Pro Tyr Ser Tyr Ser Arg Glu Tyr Asn Glu Tyr
 85 90 95
 Glu Asn Ile Lys Leu Glu Arg His Val Val Ser Phe Asp Ser Ser Arg
 100 105 110
 Pro Thr Ser Gly Lys Met Asn Cys Asp Val Cys Gly Leu Ser Cys Ile
 115 120 125
 Ser Phe Asn Val Leu Met Val His Lys Arg Ser His Thr Gly Glu Arg
 130 135 140
 Pro Phe Gln Cys Asn Gln Cys Gly Ala Ser Phe Thr Gln Lys Gly Asn
 145 150 155 160
 Leu Leu Arg His Ile Lys Leu His Thr Gly Glu Lys Pro Phe Lys Cys
 165 170 175
 His Leu Cys Asn Tyr Ala Cys Gln Arg Asp Ala Leu Thr Gly His
 180 185 190
 Leu Arg Thr His Ser Val Glu Lys Pro Tyr Lys Cys Glu Phe Cys Gly
 195 200 205

Arg Ser Tyr Lys Gln Arg Ser Ser Leu Glu Glu His Lys Glu Arg Cys
 210 215 220
 Arg Thr Phe Leu Gln Ser Thr Asp Pro Gly Asp Thr Ala Ser Ala Glu
 225 230 235 240
 Ala Arg His Ile Lys Ala Glu Met Gly Ser Glu Arg Ala Leu Val Leu
 245 250 255
 Asp Arg Leu Ala Ser Asn Val Ala Lys Arg Lys Ser Ser Met Pro Gln
 260 265 270
 Lys Phe Ile Gly Glu Lys Arg His Cys Phe Asp Val Asn Tyr Asn Ser
 275 280 285
 Ser Tyr Met Tyr Glu Lys Glu Ser Glu Leu Ile Gln Thr Arg Met Met
 290 295 300
 Asp Gln Ala Ile Asn Asn Ala Ile Ser Tyr Leu Gly Ala Glu Ala Leu
 305 310 315 320
 Cys Pro Leu Val Gln Thr Pro Pro Ala Pro Thr Ser Glu Met Val Pro
 325 330 335
 Val Ile Ser Ser Met Tyr Pro Ile Ala Leu Thr Arg Ala Glu Met Ser
 340 345 350
 Asn Gly Ala Pro Gln Glu Leu Glu Arg Lys Ser Ile Leu Leu Pro Glu
 355 360 365
 Lys Ser Val Pro Ser Glu Arg Gly Leu Ser Pro Asn Asn Ser Gly His
 370 375 380
 Asp Ser Thr Asp Thr Asp Ser Asn His Glu Glu Arg Gln Asn His Ile
 385 390 395 400
 Tyr Gln Gln Asn His Met Val Leu Ser Arg Ala Arg Asn Gly Met Pro
 405 410 415
 Leu Leu Lys Glu Val Pro Arg Ser Tyr Glu Leu Leu Lys Pro Pro Pro
 420 425 430
 Ile Cys Pro Arg Asp Ser Val Lys Val Ile Asp Lys Glu Gly Glu Val
 435 440 445
 Met Asp Val Tyr Arg Cys Asp His Cys Arg Val Leu Phe Leu Asp Tyr
 450 455 460
 Val Met Phe Thr Ile His Met Gly Cys His Gly Phe Arg Asp Pro Phe
 465 470 475 480
 Glu Cys Asn Met Cys Gly Asp Arg Ser His Asp Arg Tyr Glu Phe Ser
 485 490 495
 Ser His Ile Ala Arg Gly Glu His Arg Ser Leu Leu Lys
 500 505

<210> 81

<211> 440

<212> PRT

<213> Homo sapiens

<400> 81
 Met Pro Ile Pro Pro Pro Pro Pro Pro Pro Gly Pro Pro Pro Pro
 1 5 10 15
 Pro Thr Phe His Gln Ala Asn Thr Glu Gln Pro Lys Leu Ser Arg Asp
 20 25 30
 Glu Gln Arg Gly Arg Gly Ala Leu Leu Gln Asp Ile Cys Lys Gly Thr
 35 40 45
 Lys Leu Lys Lys Val Thr Asn Ile Asn Asp Arg Ser Ala Pro Ile Leu
 50 55 60
 Glu Lys Pro Lys Gly Ser Ser Gly Gly Tyr Gly Ser Gly Gly Ala Ala
 65 70 75 80
 Leu Gln Pro Lys Gly Leu Phe Gln Gly Gly Val Leu Lys Leu Arg
 85 90 95
 Pro Val Gly Ala Lys Asp Gly Ser Glu Asn Leu Ala Gly Lys Pro Ala
 100 105 110
 Leu Gln Ile Pro Ser Ser Arg Ala Ala Ala Pro Arg Pro Pro Val Ser
 115 120 125

Ala Ala Ser Gly Arg Pro Gln Asp Asp Thr Asp Ser Ser Arg Ala Ser
 130 135 140
 Leu Pro Glu Leu Pro Arg Met Gln Arg Pro Ser Leu Pro Asp Leu Ser
 145 150 155 160
 Arg Pro Asn Thr Thr Ser Ser Thr Gly Met Lys His Ser Ser Ser Ala
 165 170 175
 Pro Pro Pro Pro Pro Pro Gly Arg Arg Ala Asn Ala Pro Pro Thr Pro
 180 185 190
 Leu Pro Met His Ser Ser Lys Ala Pro Ala Tyr Asn Arg Glu Lys Pro
 195 200 205
 Leu Pro Pro Thr Pro Gly Gln Arg Leu His Pro Gly Arg Glu Gly Pro
 210 215 220
 Pro Ala Pro Pro Pro Val Lys Pro Pro Pro Ser Pro Val Asn Ile Arg
 225 230 235 240
 Thr Gly Pro Ser Gly Gln Ser Leu Ala Pro Pro Pro Pro Tyr Arg
 245 250 255
 Gln Pro Pro Gly Val Pro Asn Gly Pro Ser Ser Pro Thr Asn Glu Ser
 260 265 270
 Ala Pro Glu Leu Pro Gln Arg His Asn Ser Leu His Arg Lys Thr Pro
 275 280 285
 Gly Pro Val Arg Gly Leu Ala Pro Pro Pro Pro Thr Ser Ala Ser Pro
 290 295 300
 Ser Leu Leu Ser Asn Arg Pro Pro Pro Pro Ala Arg Asp Pro Pro Ser
 305 310 315 320
 Arg Gly Ala Ala Pro Pro Pro Pro Pro Pro Val Ile Arg Asn Gly Ala
 325 330 335
 Arg Asp Ala Pro Pro Pro Pro Pro Tyr Arg Met His Gly Ser Glu
 340 345 350
 Pro Pro Ser Arg Gly Lys Pro Pro Pro Pro Pro Ser Arg Thr Pro Ala
 355 360 365
 Gly Pro Pro Pro Pro Pro Pro Pro Pro Pro Leu Arg Asn Gly His Arg Asp
 370 375 380
 Ser Ile Thr Thr Val Arg Ser Phe Leu Asp Asp Phe Glu Ser Lys Tyr
 385 390 395 400
 Ser Phe His Pro Val Glu Asp Phe Pro Ala Pro Glu Glu Tyr Lys His
 405 410 415
 Phe Gln Arg Ile Tyr Pro Ser Lys Thr Asn Arg Ala Ala Arg Gly Ala
 420 425 430
 Pro Pro Leu Pro Pro Ile Leu Arg
 435 440
 <210> 82
 <211> 205
 <212> PRT
 <213> Homo sapiens

<400> 82
 Met Ser Ile Met Ser Tyr Asn Gly Gly Ala Val Met Ala Met Lys Gly
 1 5 10 15
 Lys Asn Cys Val Ala Ile Ala Ala Asp Arg Arg Phe Gly Ile Gln Ala
 20 25 30
 Gln Met Val Thr Thr Asp Phe Gln Lys Ile Phe Pro Met Gly Asp Arg
 35 40 45
 Leu Tyr Ile Gly Leu Ala Gly Leu Ala Thr Asp Val Gln Thr Val Ala
 50 55 60
 Gln Arg Leu Lys Phe Arg Leu Asn Leu Tyr Glu Leu Lys Glu Gly Arg
 65 70 75 80
 Gln Ile Lys Pro Tyr Thr Leu Met Ser Met Val Ala Asn Leu Leu Tyr
 85 90 95
 Glu Lys Arg Phe Gly Pro Tyr Tyr Thr Glu Pro Val Ile Ala Gly Leu
 100 105 110

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Asp Pro Lys Thr Phe Lys Pro Phe Ile Cys Ser Leu Asp Leu Ile Gly
 115 120 125
 Cys Pro Met Val Thr Asp Asp Phe Val Val Ser Gly Thr Cys Ala Glu
 130 135 140
 Gln Met Tyr Gly Met Cys Glu Ser Leu Trp Glu Pro Asn Met Asp Pro
 145 150 155 160
 Asp His Leu Phe Glu Thr Ile Ser Gln Ala Met Leu Asn Ala Val Asp
 165 170 175
 Arg Asp Ala Val Ser Gly Met Gly Val Ile Val His Ile Ile Glu Lys
 180 185 190
 Asp Lys Ile Thr Thr Arg Thr Leu Lys Ala Arg Met Asp
 195 200 205

<210> 83

<211> 190

<212> PRT

<213> Homo sapiens

<400> 83
 Leu Thr Arg Ser Cys Ser Thr Cys Cys Pro Ala Val Ala Cys Leu Val
 1 5 10 15
 Gly Arg Gly Val Val Thr Ser Gly Ala Met His Gln Cys Trp Gly Glu
 20 25 30
 Glu Met Leu Gln Gly Met Leu Leu Trp Gly Trp Ala Thr Cys Pro Leu
 35 40 45
 Ser Asn Pro Gly Arg Trp Gly Arg Thr Val Gly Leu Gln His Pro Ala
 50 55 60
 Val Val Ser Ala Phe Arg Ala Leu Leu Leu Leu Met Leu Thr Val His
 65 70 75 80
 Val Ser Tyr Leu Ser Leu Ile Arg Phe Asp Tyr Gly Tyr Asn Leu Val
 85 90 95
 Ala Asn Val Ala Ile Gly Leu Val Asn Val Val Trp Trp Leu Ala Trp
 100 105 110
 Cys Leu Trp Asn Gln Arg Arg Leu Pro His Val Arg Lys Cys Val Val
 115 120 125
 Val Val Leu Leu Leu Gln Gly Leu Ser Leu Leu Glu Leu Leu Asp Phe
 130 135 140
 Pro Pro Leu Phe Trp Val Leu Asp Ala His Ala Ile Trp His Ile Ser
 145 150 155 160
 Thr Ile Pro Val His Val Leu Phe Phe Ser Phe Leu Glu Asp Asp Ser
 165 170 175
 Leu Tyr Leu Leu Lys Glu Ser Glu Asp Lys Phe Lys Leu Asp
 180 185 190

<210> 84

<211> 368

<212> PRT

<213> Homo sapiens

<400> 84
 Ala Pro Pro Pro Ala Ala Ser Gln Gly Glu Arg Met Ala Gly Leu Ala
 1 5 10 15
 Ala Arg Leu Val Leu Leu Ala Gly Ala Ala Ala Leu Ala Ser Gly Ser
 20 25 30
 Gln Gly Asp Arg Glu Pro Val Tyr Arg Asp Cys Val Leu Gln Cys Glu
 35 40 45
 Glu Gln Asn Cys Ser Gly Gly Ala Leu Asn His Phe Arg Ser Arg Gln
 50 55 60

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Pro Ile Tyr Met Ser Leu Ala Gly Trp Thr Cys Arg Asp Asp Cys Lys
 65 70 75 80
 Tyr Glu Cys Met Trp Val Thr Val Gly Leu Tyr Leu Gln Glu Gly His
 85 90 95
 Lys Val Pro Gln Phe His Gly Lys Trp Pro Phe Ser Arg Phe Leu Phe
 100 105 110
 Phe Gln Glu Pro Ala Ser Ala Val Ala Ser Phe Leu Asn Gly Leu Ala
 115 120 125
 Ser Leu Val Met Leu Cys Arg Tyr Arg Thr Phe Val Pro Ala Ser Ser
 130 135 140
 Pro Met Tyr His Thr Cys Val Ala Phe Ala Trp Val Ser Leu Asn Ala
 145 150 155 160
 Trp Phe Trp Ser Thr Val Phe His Thr Arg Asp Thr Asp Leu Thr Glu
 165 170 175
 Lys Met Asp Tyr Phe Cys Ala Ser Thr Val Ile Leu His Ser Ile Tyr
 180 185 190
 Leu Cys Cys Val Arg Thr Val Gly Leu Gln His Pro Ala Val Val Ser
 195 200 205
 Ala Phe Arg Ala Leu Leu Leu Met Leu Thr Val His Val Ser Tyr
 210 215 220
 Leu Ser Leu Ile Arg Phe Asp Tyr Gly Tyr Asn Leu Val Ala Asn Val
 225 230 235 240
 Ala Ile Gly Leu Val Asn Val Val Trp Trp Leu Ala Trp Cys Leu Trp
 245 250 255
 Asn Gln Arg Arg Leu Pro His Val Arg Lys Cys Val Val Val Leu
 260 265 270
 Leu Leu Gln Gly Leu Ser Leu Leu Glu Leu Leu Asp Phe Pro Pro Leu
 275 280 285
 Phe Trp Val Leu Asp Ala His Ala Ile Trp His Ile Ser Thr Ile Pro
 290 295 300
 Val His Val Leu Phe Phe Ser Phe Leu Glu Asp Asp Ser Leu Tyr Leu
 305 310 315 320
 Leu Lys Glu Ser Glu Asp Lys Phe Lys Leu Val Glu Ala Asp Trp Ile
 325 330 335
 Phe Ala Leu Pro Leu Thr Pro Cys Pro Ser Leu Arg Glu Gly Ser Tyr
 340 345 350
 Ala Arg Thr Pro Thr Ser Gly Thr Arg Val Ala Cys Ala Ser Phe Phe
 355 360 365
 <210> 85
 <211> 190
 <212> PRT
 <213> Homo sapiens
 <400> 85
 Leu Thr Arg Ser Cys Ser Thr Cys Cys Pro Ala Val Ala Cys Leu Val
 1 5 10 15
 Gly Arg Gly Val Val Thr Ser Gly Ala Met His Gln Cys Trp Gly Glu
 20 25 30
 Glu Met Leu Gln Gly Met Leu Leu Trp Gly Trp Ala Thr Cys Pro Leu
 35 40 45
 Ser Asn Pro Gly Arg Trp Gly Arg Thr Val Gly Leu Gln His Pro Ala
 50 55 60
 Val Val Ser Ala Phe Arg Ala Leu Leu Leu Met Leu Thr Val His
 65 70 75 80
 Val Ser Tyr Leu Ser Leu Ile Arg Phe Asp Tyr Gly Tyr Asn Leu Val
 85 90 95
 Ala Asn Val Ala Ile Gly Leu Val Asn Val Val Trp Trp Leu Ala Trp
 100 105 110
 Cys Leu Trp Asn Gln Arg Arg Leu Pro His Val Arg Lys Cys Val Val
 115 120 125

Val Val Leu Leu Leu Gln Gly Leu Ser Leu Leu Glu Leu Leu Asp Phe
 130 135 140
 Pro Pro Leu Phe Trp Val Leu Asp Ala His Ala Ile Trp His Ile Ser
 145 150 155 160
 Thr Ile Pro Val His Val Leu Phe Phe Ser Phe Leu Glu Asp Asp Ser
 165 170 175
 Leu Tyr Leu Leu Lys Glu Ser Glu Asp Lys Phe Lys Leu Asp
 180 185 190
 <210> 86

<211> 318

<212> PRT

<213> Homo sapiens

<400> 86
 Met Ala Gly Leu Ala Ala Arg Leu Val Leu Leu Ala Gly Ala Ala Ala
 1 5 10 15
 Leu Ala Ser Gly Ser Gln Gly Asp Arg Glu Pro Val Tyr Arg Asp Cys
 20 25 30
 Val Leu Gln Cys Glu Glu Gln Asn Cys Ser Gly Gly Ala Leu Asn His
 35 40 45
 Phe Arg Ser Arg Gln Pro Ile Tyr Met Ser Leu Ala Gly Trp Thr Cys
 50 55 60
 Arg Asp Asp Cys Lys Tyr Glu Cys Met Trp Val Thr Val Gly Leu Tyr
 65 70 75 80
 Leu Gln Glu Gly His Lys Val Pro Gln Phe His Gly Lys Trp Pro Phe
 85 90 95
 Ser Arg Phe Leu Phe Phe Gln Glu Pro Ala Ser Ala Val Ala Ser Phe
 100 105 110
 Leu Asn Gly Leu Ala Ser Leu Val Met Leu Cys Arg Tyr Arg Thr Phe
 115 120 125
 Val Pro Ala Ser Ser Pro Met Tyr His Thr Cys Val Ala Phe Ala Trp
 130 135 140
 Val Ser Leu Asn Ala Trp Phe Trp Ser Thr Val Phe His Thr Arg Asp
 145 150 155 160
 Thr Asp Leu Gln Arg Lys Trp Thr Thr Ser Val Pro Pro Val Ser Tyr
 165 170 175
 Thr Gln Ser Thr Cys Ala Ala Ser Gly Pro Trp Gly Cys Ser Thr Gln
 180 185 190
 Leu Trp Ser Ser Ala Phe Arg Ala Leu Leu Leu Met Leu Thr Val
 195 200 205
 His Val Ser Tyr Leu Ser Leu Ile Arg Phe Asp Tyr Gly Tyr Asn Leu
 210 215 220
 Val Ala Asn Val Ala Ile Gly Leu Val Asn Val Val Trp Trp Leu Ala
 225 230 235 240
 Trp Cys Leu Trp Asn Gln Arg Arg Leu Pro His Val Arg Lys Cys Val
 245 250 255
 Val Val Val Leu Leu Leu Gln Gly Leu Ser Leu Leu Glu Leu Asp
 260 265 270
 Phe Pro Pro Leu Phe Trp Val Leu Asp Ala His Ala Ile Trp His Ile
 275 280 285
 Ser Thr Ile Pro Val His Val Leu Phe Phe Ser Phe Leu Glu Asp Asp
 290 295 300
 Ser Leu Tyr Leu Leu Lys Glu Ser Glu Asp Lys Phe Lys Leu
 305 310 315
 <210> 87

<211> 226

<212> PRT

<213> Homo sapiens

<400> 87
 Met Ala Gly Leu Ala Ala Arg Leu Val Leu Leu Ala Gly Ala Ala Ala
 1 5 10 15
 Leu Ala Ser Gly Ser Gln Gly Asp Arg Glu Pro Val Tyr Arg Asp Cys
 20 25 30
 Val Leu Gln Cys Glu Glu Gln Asn Cys Ser Gly Gly Ala Leu Asn His
 35 40 45
 Phe Arg Ser Arg Gln Pro Ile Tyr Met Ser Leu Ala Gly Trp Thr Cys
 50 55 60
 Arg Asp Asp Cys Lys Tyr Glu Cys Met Trp Val Thr Val Gly Leu Tyr
 65 70 75 80
 Leu Gln Glu Gly His Lys Val Pro Gln Phe His Gly Lys Trp Pro Phe
 85 90 95
 Ser Arg Phe Leu Phe Phe Gln Glu Pro Ala Ser Ala Val Ala Ser Phe
 100 105 110
 Leu Asn Gly Leu Ala Ser Leu Val Met Leu Cys Arg Tyr Arg Thr Phe
 115 120 125
 Val Pro Ala Ser Ser Pro Met Tyr His Thr Cys Val Ala Phe Ala Trp
 130 135 140
 Val Ser Leu Asn Ala Trp Phe Trp Ser Thr Val Phe His Thr Arg Asp
 145 150 155 160
 Thr Asp Leu Thr Glu Lys Met Asp Tyr Phe Cys Ala Ser Thr Val Ile
 165 170 175
 Leu His Ser Ile Tyr Leu Cys Cys Val Arg Pro Gly Gln Arg Gly Val
 180 185 190
 Val Ala Gly Leu Val Pro Val Glu Pro Ala Ala Ala Ala Ser Arg Ala
 195 200 205
 Gln Val Arg Gly Gly Gly Leu Ala Ala Ala Gly Ala Val Pro Ala Arg
 210 215 220
 Ala Ala
 225
 <210> 88
 <211> 320
 <212> PRT
 <213> Homo sapiens

<400> 88
 Met Ala Gly Leu Ala Ala Arg Leu Val Leu Leu Ala Gly Ala Ala Ala
 1 5 10 15
 Leu Ala Ser Gly Ser Gln Gly Asp Arg Glu Pro Val Tyr Arg Asp Cys
 20 25 30
 Val Leu Gln Cys Glu Glu Gln Asn Cys Ser Gly Gly Ala Leu Asn His
 35 40 45
 Phe Arg Ser Arg Gln Pro Ile Tyr Met Ser Leu Ala Gly Trp Thr Cys
 50 55 60
 Arg Asp Asp Cys Lys Tyr Glu Cys Met Trp Val Thr Val Gly Leu Tyr
 65 70 75 80
 Leu Gln Glu Gly His Lys Val Pro Gln Phe His Gly Lys Trp Pro Phe
 85 90 95
 Ser Arg Phe Leu Phe Phe Gln Glu Pro Ala Ser Ala Val Ala Ser Phe
 100 105 110
 Leu Asn Gly Leu Ala Ser Leu Val Met Leu Cys Arg Tyr Arg Thr Phe
 115 120 125
 Val Pro Ala Ser Ser Pro Met Tyr His Thr Cys Val Ala Phe Ala Trp
 130 135 140
 Val Ser Leu Asn Ala Trp Phe Trp Ser Thr Val Phe His Thr Arg Asp
 145 150 155 160

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Thr Asp Leu Thr Glu Lys Met Asp Tyr Phe Cys Ala Ser Thr Val Ile
 165 170 175
 Leu His Ser Ile Tyr Leu Cys Cys Val Arg Thr Val Gly Leu Gln His
 180 185 190
 Pro Ala Val Val Ser Ala Phe Arg Ala Leu Leu Leu Leu Met Leu Thr
 195 200 205
 Val His Val Ser Tyr Leu Ser Leu Ile Arg Phe Asp Tyr Gly Tyr Asn
 210 215 220
 Leu Val Ala Asn Val Ala Ile Gly Leu Val Asn Val Val Trp Trp Leu
 225 230 235 240
 Ala Trp Cys Leu Trp Asn Gln Arg Arg Leu Pro His Val Arg Lys Cys
 245 250 255
 Val Val Val Val Leu Leu Leu Gln Gly Leu Ser Leu Leu Glu Leu Leu
 260 265 270
 Asp Phe Pro Pro Leu Phe Trp Val Leu Asp Ala His Ala Ile Trp His
 275 280 285
 Ile Ser Thr Ile Pro Val His Val Leu Phe Phe Ser Phe Leu Glu Asp
 290 295 300
 Asp Ser Leu Tyr Leu Leu Lys Glu Ser Glu Asp Lys Phe Lys Leu Asp
 305 310 315 320
 <210> 89

<211> 217

<212> PRT

<213> Homo sapiens

<400> 89
 Ala Pro Pro Pro Ala Ala Ser Gln Gly Glu Arg Met Ala Gly Leu Ala
 1 5 10 15
 Ala Arg Leu Val Leu Leu Ala Gly Ala Ala Ala Leu Ala Ser Gly Ser
 20 25 30
 Gln Gly Asp Arg Glu Pro Val Tyr Arg Asp Cys Val Leu Gln Cys Glu
 35 40 45
 Glu Gln Asn Cys Ser Gly Gly Ala Leu Asn His Phe Arg Ser Arg Gln
 50 55 60
 Pro Ile Tyr Met Ser Leu Ala Gly Trp Thr Cys Arg Asp Asp Cys Lys
 65 70 75 80
 Tyr Glu Cys Met Trp Val Thr Val Gly Leu Tyr Leu Gln Glu Gly His
 85 90 95
 Lys Val Pro Gln Phe His Gly Lys Trp Pro Phe Ser Arg Phe Leu Phe
 100 105 110
 Phe Gln Glu Pro Ala Ser Ala Val Ala Ser Phe Leu Asn Gly Leu Ala
 115 120 125
 Ser Leu Val Met Leu Cys Arg Tyr Arg Thr Phe Val Pro Ala Ser Ser
 130 135 140
 Pro Met Tyr His Thr Cys Val Ala Phe Ala Trp Val Ser Leu Asn Ala
 145 150 155 160
 Trp Phe Trp Ser Thr Val Phe His Thr Arg Asp Thr Asp Leu Thr Glu
 165 170 175
 Lys Met Asp Tyr Phe Cys Ala Ser Thr Val Ile Leu His Ser Ile Tyr
 180 185 190
 Leu Cys Cys Val Ser Phe Leu Glu Asp Asp Ser Leu Tyr Leu Leu Lys
 195 200 205
 Glu Ser Glu Asp Lys Phe Lys Leu Asp
 210 215
 <210> 90

<211> 153

<212> PRT

<213> Homo sapiens

<400> 90
 Met Asn Val Gly Thr Ala His Ser Glu Val Asn Pro Asn Thr Arg Val
 1 5 10 15
 Met Asn Ser Arg Gly Ile Trp Leu Ser Tyr Val Leu Ala Ile Gly Leu
 20 25 30
 Leu His Ile Val Leu Leu Ser Ile Pro Phe Val Ser Val Pro Val Val
 35 40 45
 Trp Thr Leu Thr Asn Leu Ile His Asn Met Gly Met Tyr Ile Phe Leu
 50 55 60
 His Thr Val Lys Gly Thr Pro Phe Glu Thr Pro Asp Gln Gly Lys Ala
 65 70 75 80
 Arg Leu Leu Thr His Trp Glu Gln Met Asp Tyr Gly Val Gln Phe Thr
 85 90 95
 Ala Ser Arg Lys Phe Leu Thr Ile Thr Pro Ile Val Leu Tyr Phe Leu
 100 105 110
 Thr Ser Phe Tyr Thr Lys Tyr Asp Gln Ile His Phe Val Leu Asn Thr
 115 120 125
 Val Ser Leu Met Ser Val Leu Ile Pro Lys Leu Pro Gln Leu His Gly
 130 135 140
 Val Arg Ile Phe Gly Ile Asn Lys Tyr
 145 150
 <210> 91
 <211> 436
 <212> PRT
 <213> Homo sapiens

<400> 91
 Met Arg Arg Asp Val Asn Gly Val Thr Lys Ser Arg Phe Glu Met Phe
 1 5 10 15
 Ser Asn Ser Asp Glu Ala Val Ile Asn Lys Lys Leu Pro Lys Glu Leu
 20 25 30
 Leu Leu Arg Ile Phe Ser Phe Leu Asp Val Val Thr Leu Cys Arg Cys
 35 40 45
 Ala Gln Val Ser Arg Ala Trp Asn Val Leu Ala Leu Asp Gly Ser Asn
 50 55 60
 Trp Gln Arg Ile Asp Leu Phe Asp Phe Gln Arg Asp Ile Glu Gly Arg
 65 70 75 80
 Val Val Glu Asn Ile Ser Lys Arg Cys Gly Gly Phe Leu Arg Lys Leu
 85 90 95
 Ser Leu Arg Gly Cys Leu Gly Val Gly Asp Asn Ala Leu Arg Thr Phe
 100 105 110
 Ala Gln Asn Cys Arg Asn Ile Glu Val Leu Asn Leu Asn Gly Cys Thr
 115 120 125
 Lys Thr Thr Asp Ala Thr Cys Thr Ser Leu Ser Lys Phe Cys Ser Lys
 130 135 140
 Leu Arg His Leu Asp Leu Ala Ser Cys Thr Ser Ile Thr Asn Met Ser
 145 150 155 160
 Leu Lys Ala Leu Ser Glu Gly Cys Pro Leu Leu Glu Gln Leu Asn Ile
 165 170 175
 Ser Trp Cys Asp Gln Val Thr Lys Asp Gly Ile Gln Ala Leu Val Arg
 180 185 190
 Gly Cys Gly Gly Leu Lys Ala Leu Phe Leu Lys Gly Cys Thr Gln Leu
 195 200 205
 Glu Asp Glu Ala Leu Lys Tyr Ile Gly Ala His Cys Pro Glu Leu Val
 210 215 220
 Thr Leu Asn Leu Gln Thr Cys Leu Gln Ile Thr Asp Glu Gly Leu Ile
 225 230 235 240

Thr	Ile	Cys	Arg	Gly	Cys	His	Lys	Leu	Gln	Ser	Leu	Cys	Ala	Ser	Gly
				245					250					255	
Cys	Ser	Asn	Ile	Thr	Asp	Ala	Ile	Leu	Asn	Ala	Leu	Gly	Gln	Asn	Cys
			260					265					270		
Pro	Arg	Leu	Arg	Ile	Leu	Glu	Val	Ala	Arg	Cys	Ser	Gln	Leu	Thr	Asp
		275					280					285			
Val	Gly	Phe	Thr	Thr	Leu	Ala	Arg	Asn	Cys	His	Glu	Leu	Glu	Lys	Met
	290					295					300				
Asp	Leu	Glu	Glu	Cys	Val	Gln	Ile	Thr	Asp	Ser	Thr	Leu	Ile	Gln	Leu
305					310					315					320
Ser	Ile	His	Cys	Pro	Arg	Leu	Gln	Val	Leu	Ser	Leu	Ser	His	Cys	Glu
				325					330					335	
Leu	Ile	Thr	Asp	Asp	Gly	Ile	Arg	His	Leu	Gly	Asn	Gly	Ala	Cys	Ala
			340					345					350		
His	Asp	Gln	Leu	Glu	Val	Ile	Glu	Leu	Asp	Asn	Cys	Pro	Leu	Ile	Thr
	355						360					365			
Asp	Ala	Ser	Leu	Glu	His	Leu	Lys	Ser	Cys	His	Ser	Leu	Glu	Arg	Ile
	370					375					380				
Glu	Leu	Tyr	Asp	Cys	Gln	Gln	Ile	Thr	Arg	Ala	Gly	Ile	Lys	Arg	Leu
385					390					395					400
Arg	Thr	His	Leu	Pro	Asn	Ile	Lys	Val	His	Ala	Tyr	Phe	Ala	Pro	Val
				405					410					415	
Thr	Pro	Pro	Pro	Ser	Val	Gly	Gly	Ser	Arg	Gln	Arg	Phe	Cys	Arg	Cys
			420					425					430		
Cys	Ile	Ile	Leu												
		435													

<210> 92

<211> 204

<212> PRT

<213> Homo sapiens

<400> 92

Met	Asp	Pro	Lys	Asp	Arg	Lys	Lys	Ile	Gln	Phe	Ser	Val	Pro	Ala	Pro
1				5					10					15	
Pro	Ser	Gln	Leu	Asp	Pro	Arg	Gln	Val	Glu	Met	Ile	Arg	Arg	Arg	Arg
		20					25					30			
Pro	Thr	Pro	Ala	Met	Leu	Phe	Arg	Leu	Ser	Glu	His	Ser	Ser	Pro	Glu
		35					40					45			
Glu	Glu	Ala	Ser	Pro	His	Gln	Arg	Ala	Ser	Gly	Glu	Gly	His	His	Leu
	50					55					60				
Lys	Ser	Lys	Arg	Pro	Asn	Pro	Cys	Ala	Tyr	Thr	Pro	Pro	Ser	Leu	Lys
65					70					75					80
Ala	Val	Gln	Arg	Ile	Ala	Glu	Ser	His	Leu	Gln	Ser	Ile	Ser	Asn	Leu
				85					90					95	
Asn	Glu	Asn	Gln	Ala	Ser	Glu	Glu	Glu	Asp	Glu	Leu	Gly	Glu	Leu	Arg
		100						105					110		
Glu	Leu	Gly	Tyr	Pro	Arg	Glu	Glu	Asp	Glu	Glu	Glu	Glu	Glu	Asp	Asp
		115					120					125			
Glu	Glu	Glu	Glu	Glu	Glu	Glu	Asp	Ser	Gln	Ala	Glu	Val	Leu	Lys	Val
	130					135					140				
Ile	Arg	Gln	Ser	Ala	Gly	Gln	Lys	Thr	Thr	Cys	Gly	Gln	Gly	Leu	Glu
145					150					155					160
Gly	Pro	Trp	Glu	Arg	Pro	Pro	Pro	Leu	Asp	Glu	Ser	Glu	Arg	Asp	Gly
				165					170					175	
Gly	Ser	Glu	Asp	Gln	Val	Glu	Asp	Pro	Ala	Leu	Ser	Glu	Pro	Gly	Glu
			180					185					190		
Glu	Pro	Gln	Arg	Pro	Ser	Pro	Ser	Glu	Pro	Gly	Thr				
		195					200								

<210> 93

<211> 115

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<212> PRT

<213> Homo sapiens

<400> 93

Met	Ser	Gly	Glu	Pro	Gly	Gln	Thr	Ser	Val	Ala	Pro	Pro	Pro	Glu	Glu
1				5					10					15	
Val	Glu	Pro	Gly	Ser	Gly	Val	Arg	Ile	Val	Val	Glu	Tyr	Cys	Glu	Pro
			20					25					30		
Cys	Gly	Phe	Glu	Ala	Thr	Tyr	Leu	Glu	Leu	Ala	Ser	Ala	Val	Lys	Glu
		35					40					45			
Gln	Tyr	Pro	Gly	Ile	Glu	Ile	Glu	Ser	Arg	Leu	Gly	Gly	Thr	Gly	Ala
	50					55					60				
Phe	Glu	Ile	Glu	Ile	Asn	Gly	Gln	Leu	Val	Phe	Ser	Lys	Leu	Glu	Asn
65					70					75					80
Gly	Gly	Phe	Pro	Tyr	Glu	Lys	Asp	Leu	Ile	Glu	Ala	Ile	Arg	Arg	Ala
				85					90					95	
Ser	Asn	Gly	Glu	Thr	Leu	Glu	Lys	Ile	Thr	Asn	Ser	Arg	Pro	Pro	Cys
			100					105					110		
Val	Ile	Leu													
			115												

<210> 94

<211> 144

<212> PRT

<213> Homo sapiens

<400> 94

Met	Gly	Ala	Val	Val	Leu	Cys	Arg	Pro	Ser	Pro	Leu	Asn	Phe	Leu	Ile
1				5					10					15	
Gln	Thr	Gly	Thr	Gly	Gln	Gly	Leu	Ser	Cys	Gly	Ser	His	Met	Trp	Arg
			20					25					30		
Cys	Glu	Ala	Thr	Pro	Cys	Gly	Val	Cys	Gly	Glu	Ser	Pro	Val	Gly	Ser
		35					40					45			
Leu	Leu	Lys	Gln	His	Arg	Gly	Arg	Gly	Lys	Thr	Trp	Pro	Val	Gly	Thr
	50					55					60				
Val	Ser	Ala	Cys	Arg	Glu	Glu	Ser	Glu	Ala	Gly	Ser	Leu	Ser	Leu	Gly
65					70					75					80
Trp	Ser	Leu	Leu	Pro	Ser	Pro	Val	Gly	Leu	Gly	Ala	Val	Leu	Ile	Leu
				85					90					95	
Lys	Arg	Cys	Gly	Ser	Leu	Cys	Pro	Leu	Pro	Gly	Val	Gln	Gly	Asn	Arg
			100					105					110		
Arg	Gly	His	Trp	Ala	Cys	Phe	Leu	Pro	Pro	Asp	Pro	Ala	Ser	Pro	Thr
		115					120					125			
Pro	Cys	Ile	Ile	Gly	Asn	Phe	His	Leu	Lys	Ile	Phe	Leu	Ser	Lys	Val
	130					135						140			

<210> 95

<211> 425

<212> PRT

<213> Homo sapiens

<400> 95

Met	Gly	Gly	Gly	Asp	Leu	Asn	Leu	Lys	Lys	Ser	Trp	His	Pro	Gln	Thr
1				5					10					15	

Leu Arg Asn Val Glu Lys Val Trp Lys Ala Glu Gln Lys His Glu Ala
 20 25 30
 Glu Arg Lys Lys Ile Glu Glu Leu Gln Arg Glu Leu Arg Glu Glu Arg
 35 40 45
 Ala Arg Glu Glu Met Gln Arg Tyr Ala Glu Asp Val Gly Ala Val Lys
 50 55 60
 Lys Lys Glu Glu Lys Leu Asp Trp Met Tyr Gln Gly Pro Gly Gly Met
 65 70 75 80
 Val Asn Arg Asp Glu Tyr Leu Leu Gly Arg Pro Ile Asp Lys Tyr Val
 85 90 95
 Phe Glu Lys Met Glu Glu Lys Glu Ala Gly Cys Ser Ser Glu Thr Gly
 100 105 110
 Leu Leu Pro Gly Ser Ile Phe Ala Pro Ser Gly Ala Asn Ser Leu Leu
 115 120 125
 Asp Met Ala Ser Lys Ile Arg Glu Asp Pro Leu Phe Ile Ile Arg Lys
 130 135 140
 Lys Glu Glu Glu Lys Lys Arg Glu Val Leu Asn Asn Pro Val Lys Met
 145 150 155 160
 Lys Lys Ile Lys Glu Leu Leu Gln Met Ser Leu Glu Lys Lys Glu Lys
 165 170 175
 Lys Lys Lys Lys Glu Lys Lys Lys Lys His Lys Lys His Lys His Arg
 180 185 190
 Ser Ser Ser Ser Asp Arg Ser Ser Ser Glu Asp Glu His Ser Ala Gly
 195 200 205
 Arg Ser Gln Lys Lys Met Ala Asn Ser Ser Pro Val Leu Ser Lys Val
 210 215 220
 Pro Gly Tyr Gly Leu Gln Val Arg Asn Ser Asp Arg Asn Gln Gly Leu
 225 230 235 240
 Gln Gly Pro Leu Thr Ala Glu Gln Lys Arg Gly His Gly Met Lys Asn
 245 250 255
 His Ser Arg Ser Arg Ser Ser Ser His Ser Pro Pro Arg His Ala Ser
 260 265 270
 Lys Lys Ser Thr Arg Glu Ala Gly Ser Arg Asp Arg Arg Ser Arg Ser
 275 280 285
 Leu Gly Arg Arg Ser Arg Ser Pro Arg Pro Ser Lys Leu His Asn Ser
 290 295 300
 Lys Val Asn Arg Arg Glu Thr Gly Gln Thr Arg Ser Pro Ser Pro Lys
 305 310 315 320
 Lys Glu Val Tyr Gln Arg Arg His Ala Pro Gly Tyr Thr Arg Lys Leu
 325 330 335
 Ser Ala Glu Glu Leu Glu Arg Lys Arg Gln Glu Met Met Glu Asn Ala
 340 345 350
 Lys Trp Arg Glu Glu Glu Arg Leu Asn Ile Leu Lys Arg His Ala Lys
 355 360 365
 Asp Glu Glu Arg Glu Gln Arg Leu Glu Lys Leu Asp Ser Arg Asp Gly
 370 375 380
 Lys Phe Ile His Arg Met Lys Leu Glu Ser Ala Ser Thr Ser Ser Leu
 385 390 395 400
 Glu Asp Arg Val Lys Arg Asn Ile Tyr Ser Leu Gln Arg Thr Ser Val
 405 410 415
 Ala Leu Glu Lys Asn Phe Met Lys Arg
 420 425

<210> 96

<211> 394

<212> PRT

<213> Homo sapiens

<400> 96

Met Phe Ser Val Phe Glu Glu Ile Thr Arg Ile Val Val Lys Glu Met
 1 5 10 15

Asp	Ala	Gly	Gly	Asp	Met	Ile	Ala	Val	Arg	Ser	Leu	Val	Asp	Ala	Asp
			20					25					30		
Arg	Phe	Arg	Cys	Phe	His	Leu	Val	Gly	Glu	Lys	Arg	Thr	Phe	Phe	Gly
		35					40					45			
Cys	Arg	His	Tyr	Thr	Thr	Gly	Leu	Thr	Leu	Met	Asp	Ile	Leu	Asp	Thr
		50				55					60				
His	Gly	Asp	Lys	Trp	Leu	Asp	Glu	Leu	Asp	Ser	Gly	Leu	Gln	Gly	Gln
65					70					75					80
Lys	Ala	Glu	Phe	Gln	Ile	Leu	Asp	Asn	Val	Asp	Ser	Thr	Gly	Glu	Leu
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Thr	Leu	Glu	Thr	Val	Lys	Glu	Glu	Thr	Leu	Lys	Ser	Asp	Arg	Gln	Tyr
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Lys	Phe	Trp	Ser	Gln	Ile	Ser	Gln	Gly	His	Leu	Ser	Tyr	Lys	His	Lys
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Glu	Leu	Ser	Glu	Glu	Gln	Gln	Phe	Val	Ala	Glu	Ala	Leu	Glu	Lys	Gly
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Gly Ala Gly His Ile Ile Lys Asp Leu Tyr Leu Leu Ile Met Lys Asp
35      40      45

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 Pro Ser Pro Gly Glu Lys Ala Leu Gly Thr Pro Glu Asp Leu Asp Ser
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 Tyr Ile Asp Phe Ser Leu Glu Ser Leu Asn Gln Met Ile Leu Glu Leu
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- 105 -

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<210> 318

<211> 3123

<212> DNA

<213> Homo sapiens

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<210> 319

<211> 1817

<212> DNA

<213> Homo sapiens

<400> 319

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<210> 320

<211> 1474

<212> DNA

<213> Homo sapiens

<400> 320

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<210> 321

<211> 754

<212> DNA

<213> Homo sapiens

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<211> 749

<212> DNA

<213> Homo sapiens

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<210> 323

<211> 440

<212> DNA

<213> Homo sapiens

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<210> 324

<211> 614

<212> DNA

<213> Homo sapiens

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<210> 325

<211> 1193

<212> DNA

<213> Homo sapiens

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<210> 326

<211> 986

<212> DNA

<213> Homo sapiens

<400> 326

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<211> 2837

<212> DNA

<213> Homo sapiens

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<212> DNA

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<211> 2218

<212> DNA

<213> Homo sapiens

<400> 375

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<211> 1986

<212> DNA

<213> Homo sapiens

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<211> 2222

<212> DNA

<213> Homo sapiens

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<211> 2270

<212> DNA

<213> Homo sapiens

<400> 378

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<211> 2301

<212> DNA

<213> Homo sapiens

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<213> Homo sapiens

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<212> DNA

<213> Homo sapiens

<400> 387

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<213> Homo sapiens

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cgccctgttc	tgaaggcacc	tcctcacctc	agaaactggg	gggtgctctca	gggcaaaatc	1920
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ccgggcagca	aagcctccat	ctggaagtct	gtctgccttt	gttccttgaa	gaatgcagca	2040
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aaaagggtcc	cagatccctt	ggccctttcc	tccgaggact	tctatcctcc	ccaggccttt	2160
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<210> 391

<211> 3291

<212> DNA

<213> Homo sapiens

<400> 391

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tactcgacac	gcctcctgcc	tgtggacatt	cgacagtact	tggctgtctg	gattgaagac	180
cagaactggc	aggaagctgc	acttgggagt	gatgattcca	aggctaccat	gctattcttc	240
cacttcttgg	atcagctgaa	ctatgagtgt	ggcgttgca	gccaggacc	agagtccttg	300
ttgctgcagc	acaatttgcg	gaaattctgc	cgggacattc	agcccttttc	ccaggatcct	360
accagttgg	ctgagatgat	ctttaacctc	cttctggaag	aaaaaagaat	tttgatccag	420
gctcagaggg	cccaattgga	acaaggagag	ccagttctcg	aaacacctgt	ggagagccag	480
caacatgaga	ttgaatcccg	gatcctggat	ttaagggcta	tgatggagaa	gctggtaaaa	540
tccatcagcc	aactgaaaga	ccagcaggat	gtcttctgct	tccgatataa	gatccaggcc	600
aaaggggaaga	caccctctct	ggacccccat	cagaccaaag	agcagaagat	tctgcaggaa	660
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gcccagggtca	cagagtttgt	acagcgtctg	ctccacagag	cctttgtggg	agaaacccag	1020
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aaccagaaaa	ccttgacccc	cgagaagggg	cagagtcagg	gtttgatttg	ggactttggg	1260
tacctgactc	tgggtggagca	acgttcagg	gggttcaggaa	agggcagcaa	taaggggcca	1320
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aacaagctgt	tggggcagaa	ctgtaggact	gaggatccat	tattgtcctg	ggctgacttc	1680
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gagttgggtac	atgaccacct	gaaggatctc	tggaaatgatg	gacgcatcat	gggctttgtg	1800
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aaggtgctca	tctactctgt	gcaaccgtac	acgaaggagg	tgtctgcagtc	actcccgtg	1980
actgaaatca	tccgccatta	ccagttgtct	actgaggaga	atatacctga	aaacccactg	2040
cgcttctctc	atccccgaat	cccccggtat	gaagcttttg	ggtgctacta	ccaggagaaa	2100
gttaattctcc	aggaacggag	gaaataacctg	aaacacaggc	tcattgtggg	ctctaataga	2160
caggtggatg	aactgcaaca	accgctggag	cttaagccag	agccagagct	ggagtcatta	2220
gagctggaac	tagggctggg	gccagagcca	gagctcagcc	tggacttaga	gccactgctg	2280
aaggcagggc	tggatctggg	gccagagcta	gagtctgtgc	tggagtccac	tctggagcct	2340
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[illegible]

<211> 1283

<212> DNA

<213> Homo sapiens

<400>	392						
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cgctctggta	ccagggtctgc	ttttaactct	ggtaaagtgg	atattgttgc	catcaatgac		180
cccttcattg	acctgaacta	catggtttac	atgttccaat	atgattccac	ccatggcaaa		240
ttccatggca	ccgtcaaggc	tgagaaacgg	aagcttgtca	tcaattggaaa	tcccatcacc		300
atcttccagg	agcgagatcc	ctccaaaatc	aagtggggcg	atgctggcgc	tgagtacgtc		360
gtggagtcga	ctggcgctct	caccaccatg	gagaaggctg	gggctcattt	gcagggggga		420
gccaaaaggg	tcatactctc	tgccccctct	gctgatgccc	ccatgttcgt	catgggtgtg		480
aacctatgaga	agtatgacaa	cagcctcaag	atcatcagca	atgcctcctg	caccaccaac		540
tgcttagcac	ccctggccaa	ggctatccat	gacaactttg	gtatcgtgga	aggactcatg		600
accacagtcc	atgccatcac	tgccaccag	aagactgtgg	atggcccttc	cgggaaactg		660
tggcgtgatg	gccgcggggc	tctccagaac	atcatccctg	cctctactgg	cgctgccaa		720
gctgtggggc	aggctcatccc	tgagctgaac	gggaagctca	ctggcatggc	cttccgtgtc		780
cccatggcca	acgtgtcagt	ggtagacctg	acctgccgtc	tagaaaaacc	tgccaaatat		840
gatgacatca	agaaggtggt	gaagcaggcg	tcggaggggc	ccctcaaggg	catcctgggc		900
tacactgagc	accaggtggt	ctcctctgac	ttcaacagcg	acacccactc	ctccaccttt		960
gacgctgggg	ctggcatatgc	cctcaacgac	cactttgtca	agctcatttc	ctggtatgac		1020
aacgaatttg	gctacagcaa	cagggtggtg	gacctcatgg	cccacatggc	ctccaaggag		1080
taagaccctt	ggaccaccag	ccccagcaag	agcacaagag	gaagagagag	acctctactg		1140
ctgggggagtc	cctgccacac	tcagtcccc	accacactga	atctcccttc	ctcacagttg		1200
ccatgtagac	cccttgaaga	ggggaggggc	ctagggagcc	gcaccttgtc	atgtaccatc		1260
aataaagtac	cctgtgctca	acc					1283
<210>	393						

<211> 331

<212> PRT

<213> Homo sapiens

<400> 393															
Met	Gly	Gly	Ser	Ala	Gly	Arg	Glu	Leu	Asp	Ala	Gly	Arg	Lys	Pro	Lys
1			5						10					15	
Leu	Thr	Arg	Thr	Gln	Ser	Ala	Phe	Ser	Pro	Val	Ser	Phe	Ser	Pro	Leu
			20					25					30		
Phe	Thr	Gly	Glu	Thr	Val	Ser	Leu	Val	Asp	Val	Asp	Ile	Ser	Gln	Arg
		35					40					45			

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Gly	Leu	Thr	Ser	Pro	His	Pro	Pro	Thr	Pro	Pro	Pro	Pro	Pro	Arg	Arg
50						55				60					
Ser	Leu	Ser	Leu	Leu	Asp	Ile	Ser	Gly	Thr	Leu	Pro	Thr	Ser	Val	
65					70				75					80	
Leu	Val	Ala	Pro	Met	Gly	Ser	Ser	Leu	Gln	Ser	Phe	Pro	Leu	Pro	Pro
				85					90					95	
Pro	Pro	Pro	Pro	His	Ala	Pro	Asp	Ala	Phe	Pro	Arg	Ile	Ala	Pro	Ile
				100				105					110		
Arg	Ala	Ala	Glu	Ser	Leu	His	Ser	Gln	Pro	Pro	Gln	His	Leu	Gln	Cys
				115				120				125			
Pro	Leu	Tyr	Arg	Pro	Asp	Ser	Ser	Ser	Phe	Ala	Ala	Ser	Leu	Arg	Glu
				130				135				140			
Leu	Glu	Lys	Cys	Gly	Trp	Tyr	Trp	Gly	Pro	Met	Asn	Trp	Glu	Asp	Ala
145					150					155					160
Glu	Met	Lys	Leu	Lys	Gly	Lys	Pro	Asp	Gly	Ser	Phe	Leu	Val	Arg	Asp
				165					170					175	
Ser	Ser	Asp	Pro	Arg	Tyr	Ile	Leu	Ser	Leu	Ser	Phe	Arg	Ser	Gln	Gly
				180				185					190		
Ile	Thr	His	His	Thr	Arg	Met	Glu	His	Tyr	Arg	Gly	Thr	Phe	Ser	Leu
				195			200					205			
Trp	Cys	His	Pro	Lys	Phe	Glu	Asp	Arg	Cys	Gln	Ser	Val	Val	Glu	Phe
				210			215					220			
Ile	Lys	Arg	Ala	Ile	Met	His	Ser	Lys	Asn	Gly	Lys	Phe	Leu	Tyr	Phe
225					230					235					240
Leu	Arg	Ser	Arg	Val	Pro	Gly	Leu	Pro	Pro	Thr	Pro	Val	Gln	Leu	Leu
				245						250				255	
Tyr	Pro	Val	Ser	Arg	Phe	Ser	Asn	Val	Lys	Ser	Leu	Gln	His	Leu	Cys
				260				265					270		
Arg	Phe	Arg	Ile	Arg	Gln	Leu	Val	Arg	Ile	Asp	His	Ile	Pro	Asp	Leu
				275				280				285			
Pro	Leu	Pro	Lys	Pro	Leu	Ile	Ser	Tyr	Ile	Arg	Lys	Phe	Tyr	Tyr	Tyr
				290				295				300			
Asp	Pro	Gln	Glu	Glu	Val	Tyr	Leu	Ser	Leu	Lys	Glu	Ala	Gln	Leu	Ile
305					310					315					320
Ser	Lys	Gln	Lys	Gln	Glu	Val	Glu	Pro	Ser	Thr					
				325					330						

<210> 394

<211> 306

<212> PRT

<213> Homo sapiens

<400> 394

Met	Ala	Ala	Pro	Ile	Pro	Gln	Gly	Phe	Ser	Cys	Leu	Ser	Arg	Val	Leu
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Gly	Trp	Trp	Ser	Arg	Gln	Pro	Val	Leu	Val	Thr	Gln	Ser	Ala	Ala	Ile
			20					25					30		
Val	Pro	Val	Arg	Thr	Lys	Lys	Arg	Phe	Thr	Pro	Pro	Ile	Tyr	Gln	Pro
				35			40					45			
Lys	Phe	Lys	Thr	Glu	Lys	Glu	Phe	Met	Gln	His	Ala	Arg	Lys	Ala	Gly
				50			55				60				
Leu	Val	Ile	Pro	Pro	Glu	Lys	Ser	Asp	Arg	Ser	Ile	His	Leu	Ala	Cys
65					70					75					80
Thr	Ala	Gly	Ile	Phe	Asp	Ala	Tyr	Val	Pro	Pro	Glu	Gly	Asp	Ala	Arg
				85					90					95	
Ile	Ser	Ser	Leu	Ser	Lys	Glu	Gly	Leu	Ile	Glu	Arg	Thr	Glu	Arg	Met
				100				105					110		
Lys	Lys	Thr	Met	Ala	Ser	Gln	Val	Ser	Ile	Arg	Arg	Ile	Lys	Asp	Tyr
				115				120				125			
Asp	Ala	Asn	Phe	Lys	Ile	Lys	Asp	Phe	Pro	Glu	Lys	Ala	Lys	Asp	Ile
				130			135					140			

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Phe Ile Glu Ala His Leu Cys Leu Asn Asn Ser Asp His Asp Arg Leu
 145 150 155 160
 His Thr Leu Val Thr Glu His Cys Phe Pro Asp Met Thr Trp Asp Ile
 165 170 175
 Lys Tyr Lys Thr Val Arg Trp Ser Phe Val Glu Ser Leu Glu Pro Ser
 180 185 190
 His Val Val Gln Val Arg Cys Ser Ser Met Met Asn Gln Gly Asn Val
 195 200 205
 Tyr Gly Gln Ile Thr Val Arg Met His Thr Arg Gln Thr Leu Ala Ile
 210 215 220
 Tyr Asp Arg Phe Gly Arg Leu Met Tyr Gly Gln Glu Asp Val Pro Lys
 225 230 235 240
 Asp Val Leu Glu Tyr Val Val Phe Glu Lys Gln Leu Thr Asn Pro Tyr
 245 250 255
 Gly Ser Trp Arg Met His Thr Lys Ile Val Pro Pro Trp Ala Pro Pro
 260 265 270
 Lys Gln Pro Ile Leu Lys Thr Val Met Ile Pro Gly Pro Gln Leu Lys
 275 280 285
 Pro Glu Glu Tyr Glu Glu Ala Gln Gly Glu Ala Gln Lys Pro Gln
 290 295 300
 Leu Ala
 305
 <210> 395
 <211> 557
 <212> PRT
 <213> Homo sapiens

<400> 395
 Met Val Ser Lys Leu Thr Ser Leu Gln Gln Glu Leu Leu Ser Ala Leu
 1 5 10 15
 Leu Ser Ser Gly Val Thr Lys Glu Val Leu Val Gln Ala Leu Glu Glu
 20 25 30
 Leu Leu Pro Ser Pro Asn Phe Gly Val Lys Leu Glu Thr Leu Pro Leu
 35 40 45
 Ser Pro Gly Ser Gly Ala Glu Pro Asp Thr Lys Pro Val Phe His Thr
 50 55 60
 Leu Thr Asn Gly His Ala Lys Gly Arg Leu Ser Gly Asp Glu Gly Ser
 65 70 75 80
 Glu Asp Gly Asp Asp Tyr Asp Thr Pro Pro Ile Leu Lys Glu Leu Gln
 85 90 95
 Ala Leu Asn Thr Glu Glu Ala Ala Glu Gln Arg Ala Glu Val Asp Arg
 100 105 110
 Met Leu Ser Glu Asp Pro Trp Arg Ala Ala Lys Met Ile Lys Gly Tyr
 115 120 125
 Met Gln Gln His Asn Ile Pro Gln Arg Glu Val Val Asp Val Thr Gly
 130 135 140
 Leu Asn Gln Ser His Leu Ser Gln His Leu Asn Lys Gly Thr Pro Met
 145 150 155 160
 Lys Thr Gln Lys Arg Ala Ala Leu Tyr Thr Trp Tyr Val Arg Lys Gln
 165 170 175
 Arg Glu Ile Leu Arg Gln Phe Asn Gln Thr Val Gln Ser Ser Gly Asn
 180 185 190
 Met Thr Asp Lys Ser Ser Gln Asp Gln Leu Leu Phe Leu Phe Pro Glu
 195 200 205
 Phe Ser Gln Gln Ser His Gly Pro Gly Gln Ser Asp Ala Cys Ser
 210 215 220
 Glu Pro Thr Asn Lys Lys Met Arg Arg Asn Arg Phe Lys Trp Gly Pro
 225 230 235 240
 Ala Ser Gln Gln Ile Leu Tyr Gln Ala Tyr Asp Arg Gln Lys Asn Pro
 245 250 255

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Ser Lys Glu Glu Arg Glu Ala Leu Val Glu Glu Cys Asn Arg Ala Glu
 260 265 270
 Cys Leu Gln Arg Gly Val Ser Pro Ser Lys Ala His Gly Leu Gly Ser
 275 280 285
 Asn Leu Val Thr Glu Val Arg Val Tyr Asn Trp Phe Ala Asn Arg Arg
 290 295 300
 Lys Glu Glu Ala Phe Arg Gln Lys Leu Ala Met Asp Ala Tyr Ser Ser
 305 310 315 320
 Asn Gln Thr His Ser Leu Asn Pro Leu Leu Ser His Gly Ser Pro His
 325 330 335
 His Gln Pro Ser Ser Ser Pro Pro Asn Lys Leu Ser Gly Val Arg Tyr
 340 345 350
 Ser Gln Gln Gly Asn Asn Glu Ile Thr Ser Ser Ser Thr Ile Ser His
 355 360 365
 His Gly Asn Ser Ala Met Val Thr Ser Gln Ser Val Leu Gln Gln Val
 370 375 380
 Ser Pro Ala Ser Leu Asp Pro Gly His Asn Leu Leu Ser Pro Asp Gly
 385 390 395 400
 Lys Met Ile Ser Val Ser Gly Gly Gly Leu Pro Pro Val Ser Thr Leu
 405 410 415
 Thr Asn Ile His Ser Leu Ser His His Asn Pro Gln Gln Ser Gln Asn
 420 425 430
 Leu Ile Met Thr Pro Leu Ser Gly Val Met Ala Ile Ala Gln Ser Leu
 435 440 445
 Asn Thr Ser Gln Ala Gln Ser Val Pro Val Ile Asn Ser Val Ala Gly
 450 455 460
 Ser Leu Ala Ala Leu Gln Pro Val Gln Phe Ser Gln Gln Leu His Ser
 465 470 475 480
 Pro His Gln Gln Pro Leu Met Gln Gln Ser Pro Gly Ser His Met Ala
 485 490 495
 Gln Gln Pro Phe Met Ala Ala Val Thr Gln Leu Gln Asn Ser His Met
 500 505 510
 Tyr Ala His Lys Gln Glu Pro Pro Gln Tyr Ser His Thr Ser Arg Phe
 515 520 525
 Pro Ser Ala Met Val Val Thr Asp Thr Ser Ser Ile Ser Thr Leu Thr
 530 535 540
 Asn Met Ser Ser Ser Lys Gln Cys Pro Leu Gln Ala Trp
 545 550 555
 <210> 396
 <211> 491
 <212> PRT
 <213> Homo sapiens

<400> 396
 Met Ser Ser Val Glu Ala Lys Ile Glu Asp Lys Lys Val Gln Arg Glu
 1 5 10 15
 Ser Lys Leu Thr Ser Gly Lys Leu Glu Asn Leu Arg Lys Glu Lys Ile
 20 25 30
 Asn Phe Leu Arg Asn Lys His Lys Ile His Val Gln Gly Thr Asp Leu
 35 40 45
 Pro Asp Pro Ile Ala Thr Phe Gln Gln Leu Asp Gln Glu Tyr Lys Ile
 50 55 60
 Asn Ser Arg Leu Leu Gln Asn Ile Leu Asp Ala Gly Phe Gln Met Pro
 65 70 75 80
 Thr Pro Ile Gln Met Gln Ala Ile Pro Val Met Leu His Gly Arg Glu
 85 90 95
 Leu Leu Ala Ser Ala Pro Thr Gly Ser Gly Lys Thr Leu Ala Phe Ser
 100 105 110
 Ile Pro Ile Leu Met Gln Leu Lys Gln Pro Ala Asn Lys Gly Phe Arg
 115 120 125

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Ala Leu Ile Ile Ser Pro Thr Arg Glu Leu Ala Ser Gln Ile His Arg
 130 135 140
 Glu Leu Ile Lys Ile Ser Glu Gly Thr Gly Phe Arg Ile His Met Ile
 145 150 155 160
 His Lys Ala Ala Val Ala Ala Lys Lys Phe Gly Pro Lys Ser Ser Lys
 165 170 175
 Lys Phe Asp Ile Leu Val Thr Thr Pro Asn Arg Leu Ile Tyr Leu Leu
 180 185 190
 Lys Gln Asp Pro Pro Gly Ile Asp Leu Ala Ser Val Glu Trp Leu Val
 195 200 205
 Val Asp Glu Ser Asp Lys Leu Phe Glu Asp Gly Lys Thr Gly Phe Arg
 210 215 220
 Asp Gln Leu Ala Ser Ile Phe Leu Ala Cys Thr Ser His Lys Val Arg
 225 230 235 240
 Arg Ala Met Phe Ser Ala Thr Phe Ala Tyr Asp Val Glu Gln Trp Cys
 245 250 255
 Lys Leu Asn Leu Asp Asn Val Ile Ser Val Ser Ile Gly Ala Arg Asn
 260 265 270
 Ser Ala Val Glu Thr Val Glu Gln Glu Leu Leu Phe Val Gly Ser Glu
 275 280 285
 Thr Gly Lys Leu Leu Ala Val Arg Glu Leu Val Lys Lys Gly Phe Asn
 290 295 300
 Pro Pro Val Leu Val Phe Val Gln Ser Ile Glu Arg Ala Lys Glu Leu
 305 310 315 320
 Phe His Glu Leu Ile Tyr Glu Gly Ile Asn Val Asp Val Ile His Ala
 325 330 335
 Glu Arg Thr Gln Gln Gln Arg Asp Asn Thr Val His Ser Phe Arg Ala
 340 345 350
 Gly Lys Ile Trp Val Leu Ile Cys Thr Ala Leu Leu Ala Arg Gly Ile
 355 360 365
 Asp Phe Lys Gly Val Asn Leu Val Ile Asn Tyr Asp Phe Pro Thr Ser
 370 375 380
 Ser Val Glu Tyr Ile His Arg Ile Gly Arg Thr Gly Arg Ala Gly Asn
 385 390 395 400
 Lys Gly Lys Ala Ile Thr Phe Phe Thr Glu Asp Asp Lys Pro Leu Leu
 405 410 415
 Arg Ser Val Ala Asn Val Ile Gln Gln Ala Gly Cys Pro Val Pro Glu
 420 425 430
 Tyr Ile Lys Gly Phe Gln Lys Leu Leu Ser Lys Gln Lys Lys Lys Met
 435 440 445
 Ile Lys Lys Pro Leu Glu Arg Glu Ser Ile Ser Thr Thr Pro Lys Cys
 450 455 460
 Phe Leu Glu Lys Ala Lys Asp Lys Gln Arg Lys Val Thr Gly Gln Asn
 465 470 475 480
 Ser Lys Lys Lys Val Ala Leu Glu Asp Lys Ser
 485 490

<210> 397

<211> 424

<212> PRT

<213> Homo sapiens

<400> 397

Met Asp Phe Ser Arg Arg Ser Phe His Arg Ser Leu Ser Ser Ser Leu
 1 5 10 15
 Gln Ala Pro Val Val Ser Thr Val Gly Met Gln Arg Leu Gly Thr Thr
 20 25 30
 Pro Ser Val Tyr Gly Gly Ala Gly Gly Arg Gly Ile Arg Ile Ser Asn
 35 40 45
 Ser Arg His Thr Val Asn Tyr Gly Ser Asp Leu Thr Gly Gly Gly Asp
 50 55 60

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Leu Phe Val Gly Asn Glu Lys Met Ala Met Gln Asn Leu Asn Asp Arg
 65 70 75 80
 Leu Ala Ser Tyr Leu Glu Lys Val Arg Thr Leu Glu Gln Ser Asn Ser
 85 90 95
 Lys Leu Glu Val Gln Ile Lys Gln Trp Tyr Glu Thr Asn Ala Pro Arg
 100 105 110
 Ala Gly Arg Asp Tyr Ser Ala Tyr Tyr Arg Gln Ile Glu Glu Leu Arg
 115 120 125
 Ser Gln Ile Lys Asp Ala Gln Leu Gln Asn Ala Arg Cys Val Leu Gln
 130 135 140
 Ile Asp Asn Ala Lys Leu Ala Ala Glu Asp Phe Arg Leu Lys Tyr Glu
 145 150 155 160
 Thr Glu Arg Gly Ile Arg Leu Thr Val Glu Ala Asp Leu Gln Gly Leu
 165 170 175
 Asn Lys Val Phe Asp Asp Leu Thr Leu His Lys Thr Asp Leu Glu Ile
 180 185 190
 Gln Ile Glu Glu Leu Asn Lys Asp Leu Ala Leu Leu Lys Lys Glu His
 195 200 205
 Gln Glu Glu Val Asp Gly Leu His Lys His Leu Gly Asn Thr Val Asn
 210 215 220
 Val Glu Val Asp Ala Ala Pro Gly Leu Asn Leu Gly Val Ile Met Asn
 225 230 235 240
 Glu Met Arg Gln Lys Tyr Glu Val Met Ala Gln Lys Asn Leu Gln Glu
 245 250 255
 Ala Lys Glu Gln Phe Glu Arg Gln Thr Ala Val Leu Gln Gln Gln Val
 260 265 270
 Thr Val Asn Thr Glu Glu Leu Lys Gly Thr Glu Val Gln Leu Thr Glu
 275 280 285
 Leu Arg Arg Thr Ser Gln Ser Leu Glu Ile Glu Leu Gln Ser His Leu
 290 295 300
 Ser Met Lys Glu Ser Leu Glu His Thr Leu Glu Glu Thr Lys Ala Arg
 305 310 315 320
 Tyr Ser Ser Gln Leu Ala Asn Leu Gln Ser Leu Leu Ser Ser Leu Glu
 325 330 335
 Ala Gln Leu Met Gln Ile Arg Ser Asn Met Glu Arg Gln Asn Asn Glu
 340 345 350
 Tyr His Ile Leu Leu Asp Ile Lys Thr Arg Leu Glu Gln Glu Ile Ala
 355 360 365
 Thr Tyr Arg Arg Leu Leu Glu Gly Glu Asp Val Lys Thr Thr Glu Tyr
 370 375 380
 Gln Leu Ser Thr Leu Glu Glu Arg Asp Ile Lys Lys Thr Arg Lys Ile
 385 390 395 400
 Lys Thr Val Val Gln Glu Val Val Asp Gly Lys Val Val Ser Ser Glu
 405 410 415
 Val Lys Glu Val Glu Glu Asn Ile
 420

<210> 398

<211> 209

<212> PRT

<213> Homo sapiens

<400> 398

Met Glu Lys His His Val Pro Ser Asp Phe Asn Val Asn Val Lys Val
 1 5 10 15
 Asp Thr Gly Pro Arg Glu Asp Leu Ile Lys Val Leu Glu Asp Met Arg
 20 25 30
 Gln Glu Tyr Glu Leu Ile Ile Lys Lys Lys His Arg Asp Leu Asp Thr
 35 40 45
 Trp Tyr Lys Glu Gln Ser Ala Ala Met Ser Gln Glu Ala Ala Ser Pro
 50 55 60

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Ala	Thr	Val	Gln	Ser	Arg	Gln	Gly	Asp	Ile	His	Glu	Leu	Lys	Arg	Thr
65					70					75					80
Phe	Gln	Ala	Leu	Glu	Ile	Asp	Leu	Gln	Thr	Gln	Tyr	Ser	Thr	Lys	Ser
			85						90					95	
Ala	Leu	Glu	Asn	Met	Leu	Ser	Glu	Thr	Gln	Ser	Arg	Tyr	Ser	Cys	Lys
			100					105					110		
Leu	Gln	Asp	Met	Gln	Glu	Ile	Ile	Ser	His	Tyr	Glu	Glu	Glu	Leu	Thr
		115					120					125			
Gln	Leu	Arg	His	Glu	Leu	Glu	Arg	Gln	Asn	Asn	Glu	Tyr	Gln	Val	Leu
	130					135					140				
Leu	Gly	Ile	Lys	Thr	His	Leu	Glu	Lys	Glu	Ile	Thr	Thr	Tyr	Arg	Arg
145					150					155					160
Leu	Leu	Glu	Gly	Glu	Ser	Glu	Gly	Thr	Arg	Glu	Glu	Ser	Lys	Ser	Ser
			165						170					175	
Met	Lys	Val	Ser	Ala	Thr	Pro	Lys	Ile	Lys	Ala	Ile	Thr	Gln	Glu	Thr
			180					185					190		
Ile	Asn	Gly	Arg	Leu	Val	Leu	Cys	Gln	Val	Asn	Glu	Ile	Gln	Lys	His
		195					200					205			

Ala

<210> 399

<211> 98

<212> PRT

<213> Homo sapiens

<400> 399

Met	Asp	Cys	Cys	Ala	Ser	Arg	Gly	Cys	Ser	Val	Pro	Thr	Gly	Pro	Ala
1				5					10					15	
Thr	Thr	Ile	Cys	Ser	Ser	Asp	Lys	Ser	Cys	Arg	Cys	Gly	Val	Cys	Leu
			20					25					30		
Pro	Ser	Thr	Cys	Pro	His	Thr	Val	Trp	Leu	Leu	Glu	Pro	Thr	Cys	Cys
		35				40						45			
Asp	Asn	Cys	Pro	Pro	Pro	Cys	His	Ile	Pro	Gln	Pro	Cys	Val	Pro	Thr
	50					55				60					
Cys	Phe	Leu	Leu	Asn	Ser	Cys	Gln	Pro	Thr	Pro	Gly	Leu	Glu	Thr	Leu
65				70						75					80
Asn	Leu	Thr	Thr	Phe	Thr	Gln	Pro	Cys	Cys	Glu	Pro	Cys	Leu	Pro	Arg
			85						90					95	

Gly Cys

<210> 400

<211> 98

<212> PRT

<213> Homo sapiens

<400> 400

Met	Asp	Cys	Cys	Ala	Ser	Arg	Ser	Cys	Ser	Val	Pro	Thr	Gly	Pro	Ala
1				5					10					15	
Thr	Thr	Ile	Cys	Ser	Ser	Asp	Lys	Ser	Cys	Arg	Cys	Gly	Val	Cys	Leu
			20					25					30		
Pro	Ser	Thr	Cys	Pro	His	Thr	Val	Trp	Leu	Leu	Glu	Pro	Ile	Cys	Cys
		35				40						45			
Asp	Asn	Cys	Pro	Pro	Pro	Cys	His	Ile	Pro	Gln	Pro	Cys	Val	Pro	Thr
	50					55				60					
Cys	Phe	Leu	Leu	Asn	Ser	Cys	Gln	Pro	Thr	Pro	Gly	Leu	Glu	Thr	Leu
65				70						75					80

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Asn Leu Thr Thr Phe Thr Gln Pro Cys Cys Glu Pro Cys Leu Pro Arg
 85 90 95
 Gly Cys

<210> 401

<211> 79

<212> PRT

<213> Homo sapiens

<400> 401

Met Ser Cys Cys Asp Ser Tyr Leu Gln Gly Cys Cys Ser Val Pro Thr
 1 5 10 15
 Gly Leu Ala Thr Thr Ile Cys Pro Ser Asp Ile Ser Cys Gln Cys Glu
 20 25 30
 Val Cys Leu Pro Ser Thr Cys Pro His Glu Ile Ser Leu Leu Gln Pro
 35 40 45
 Thr Cys Cys Glu Pro Gly Pro Cys Leu Ala Ala Cys Leu Thr Pro Met
 50 55 60
 Cys His Pro Val Asp Cys Ser Thr Asn Ala Thr Gln Leu Gln Pro
 65 70 75

<210> 402

<211> 98

<212> PRT

<213> Homo sapiens

<400> 402

Met Tyr Cys Cys Ala Leu Arg Ser Cys Ser Val Pro Thr Gly Pro Ala
 1 5 10 15
 Thr Thr Phe Cys Ser Phe Asp Lys Ser Cys Arg Cys Gly Val Cys Leu
 20 25 30
 Pro Ser Thr Cys Pro His Glu Ile Ser Leu Leu Gln Pro Ile Cys Cys
 35 40 45
 Asp Thr Cys Pro Pro Pro Cys Cys Lys Pro Asp Thr Tyr Val Pro Thr
 50 55 60
 Cys Trp Leu Leu Asn Asn Cys His Pro Thr Pro Gly Leu Ser Gly Ile
 65 70 75 80
 Asn Leu Thr Thr Tyr Val Gln Pro Gly Cys Glu Ser Pro Cys Glu Pro
 85 90 95
 Arg Cys

<210> 403

<211> 174

<212> PRT

<213> Homo sapiens

<400> 403

Met Thr Cys Cys Gln Thr Ser Phe Cys Gly Tyr Pro Ser Phe Ser Ile
 1 5 10 15
 Ser Gly Thr Cys Gly Ser Ser Cys Cys Gln Pro Ser Cys Cys Glu Thr
 20 25 30

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```

Ser Cys Cys Gln Pro Arg Ser Cys Gln Thr Ser Phe Cys Gly Phe Pro
    35      40      45
Ser Phe Ser Thr Ser Gly Thr Cys Ser Ser Ser Cys Cys Gln Pro Ser
    50      55      60
Cys Cys Glu Thr Ser Cys Cys Gln Pro Ser Cys Cys Glu Thr Ser Cys
    65      70      75      80
Cys Gln Pro Ser Cys Cys Gln Ile Ser Ser Cys Gly Thr Gly Cys Gly
    85      90      95
Ile Gly Gly Gly Ile Ser Tyr Gly Gln Glu Gly Ser Ser Gly Ala Val
    100      105      110
Ser Thr Arg Ile Arg Trp Cys Arg Pro Asp Ser Arg Val Glu Gly Thr
    115      120      125
Tyr Leu Pro Pro Cys Cys Val Val Ser Cys Thr Pro Pro Ser Cys Cys
    130      135      140
Gln Leu His His Ala Gln Ala Ser Cys Cys Arg Pro Ser Tyr Cys Gly
    145      150      155      160
Gln Ser Cys Cys Arg Pro Val Cys Cys Cys Glu Pro Thr Cys
    165      170

```

<210> 404

<211> 167

<212> PRT

<213> Homo sapiens

<400> 404

```

Met Thr Cys Cys Gln Thr Ser Phe Cys Gly Tyr Pro Ser Cys Ser Thr
    1      5      10      15
Ser Gly Thr Cys Gly Ser Ser Cys Cys Gln Pro Ser Cys Cys Glu Thr
    20      25      30
Ser Cys Cys Gln Pro Ser Cys Cys Gln Thr Ser Phe Cys Gly Phe Pro
    35      40      45
Ser Phe Ser Thr Ser Gly Thr Cys Ser Ser Ser Cys Cys Gln Pro Ser
    50      55      60
Cys Cys Glu Thr Ser Cys Cys Gln Pro Ser Cys Cys Gln Thr Ser Ser
    65      70      75      80
Cys Gly Thr Gly Cys Gly Ile Gly Gly Gly Ile Gly Tyr Gly Gln Glu
    85      90      95
Gly Ser Ser Gly Ala Val Ser Thr Arg Ile Arg Trp Cys Arg Pro Asp
    100      105      110
Cys Arg Val Glu Gly Thr Cys Leu Pro Pro Cys Cys Val Val Ser Cys
    115      120      125
Thr Pro Pro Thr Cys Cys Gln Leu His His Ala Glu Ala Ser Cys Cys
    130      135      140
Arg Pro Ser Tyr Cys Gly Gln Ser Cys Cys Arg Pro Val Cys Cys Cys
    145      150      155      160
Tyr Ser Cys Glu Pro Thr Cys
    165

```

<210> 405

<211> 177

<212> PRT

<213> Homo sapiens

<400> 405

```

Met Ala Cys Cys Gln Thr Ser Phe Cys Gly Phe Pro Ser Cys Ser Thr
    1      5      10      15
Ser Gly Thr Cys Gly Ser Ser Cys Cys Gln Pro Ser Cys Cys Glu Thr
    20      25      30

```

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```

Ser Ser Cys Gln Pro Arg Cys Cys Glu Thr Ser Cys Cys Gln Pro Ser
   35   40
Cys Cys Gln Thr Ser Phe Cys Gly Phe Pro Ser Phe Ser Thr Gly Gly
   50   55   60
Thr Cys Asp Ser Ser Cys Cys Gln Pro Ser Cys Cys Glu Thr Ser Cys
   65   70   75   80
Cys Gln Pro Ser Cys Tyr Gln Thr Ser Ser Cys Gly Thr Gly Cys Gly
   85   90   95
Ile Gly Gly Gly Ile Gly Tyr Gly Gln Glu Gly Ser Ser Gly Ala Val
   100  105  110
Ser Thr Arg Ile Arg Trp Cys Arg Pro Asp Cys Arg Val Glu Gly Thr
   115  120  125
Cys Leu Pro Pro Cys Cys Val Val Ser Cys Thr Pro Pro Ser Cys Cys
   130  135  140
Gln Leu His His Ala Glu Ala Ser Cys Cys Arg Pro Ser Tyr Cys Gly
   145  150  155  160
Gln Ser Cys Cys Arg Pro Val Cys Cys Cys Tyr Cys Ser Glu Pro Thr
   165  170  175
Cys

```

<210> 406

<211> 85

<212> PRT

<213> Homo sapiens

<400> 406

```

Val Thr Cys Val Pro Arg Cys Thr Arg Pro Ile Cys Glu Pro Cys Arg
   1   5  10  15
Arg Pro Val Cys Cys Asp Pro Cys Ser Leu Gln Glu Gly Cys Cys Arg
   20  25  30
Pro Ile Thr Cys Cys Pro Ser Ser Cys Thr Ala Val Val Cys Arg Pro
   35  40  45
Cys Cys Trp Ala Thr Thr Cys Cys Gln Pro Val Ser Val Gln Ser Pro
   50  55  60
Cys Cys Arg Pro Pro Cys Gly Gln Pro Thr Pro Cys Ser Thr Thr Cys
   65  70  75  80
Arg Thr Ser Ser Cys
   85

```

<210> 407

<211> 128

<212> PRT

<213> Homo sapiens

<400> 407

```

Met Thr Gly Ser Cys Cys Gly Ser Thr Leu Ser Ser Leu Ser Tyr Gly
   1   5  10  15
Gly Gly Cys Cys Gln Pro Cys Cys Cys Arg Asp Pro Cys Cys Cys Arg
   20  25  30
Pro Val Thr Cys Gln Thr Thr Val Cys Arg Pro Val Thr Cys Val Pro
   35  40  45
Arg Cys Thr Arg Pro Ile Cys Glu Pro Cys Arg Arg Pro Val Cys Cys
   50  55  60
Asp Pro Cys Ser Leu Gln Glu Gly Cys Cys Arg Pro Ile Thr Cys Cys
   65  70  75  80
Pro Ser Ser Cys Thr Ala Val Val Cys Arg Pro Cys Cys Trp Ala Thr
   85  90  95

```

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Thr	Cys	Cys	Gln	Pro	Val	Ser	Val	Gln	Ser	Pro	Cys	Cys	Arg	Pro	Pro
			100					105					110		
Cys	Gly	Gln	Pro	Thr	Pro	Cys	Ser	Thr	Thr	Cys	Arg	Thr	Ser	Ser	Cys
		115					120					125			

<210> 408

<211> 20

<212> PRT

<213> Homo sapiens

<400> 408

Met	Glu	Thr	His	Cys	Thr	Gly	Arg	Ser	Ala	Ser	Phe	Cys	Ser	Ser	Ser
1				5					10					15	
Ala	Ile	Leu	Ile												
			20												

<210> 409

<211> 210

<212> PRT

<213> Homo sapiens

<400> 409

Met	Val	Ser	Ser	Cys	Cys	Gly	Ser	Val	Cys	Ser	Asp	Gln	Gly	Cys	Gly
1				5					10					15	
Gln	Val	Leu	Cys	Gln	Glu	Thr	Cys	Cys	Arg	Pro	Ser	Cys	Cys	Gln	Thr
			20					25					30		
Thr	Cys	Cys	Arg	Thr	Thr	Cys	Tyr	Arg	Pro	Ser	Cys	Cys	Val	Ser	Ser
		35					40					45			
Cys	Cys	Arg	Pro	Gln	Cys	Cys	Gln	Ser	Val	Cys	Cys	Gln	Pro	Thr	Cys
	50					55					60				
Cys	Arg	Pro	Ser	Cys	Cys	Glu	Thr	Thr	Cys	Cys	His	Pro	Arg	Cys	Cys
65					70					75					80
Ile	Ser	Ser	Cys	Cys	Arg	Pro	Ser	Cys	Cys	Met	Ser	Ser	Cys	Cys	Lys
			85						90					95	
Pro	Gln	Cys	Cys	Gln	Ser	Val	Cys	Cys	Gln	Pro	Thr	Cys	Cys	Arg	Pro
			100					105					110		
Ser	Cys	Cys	Ile	Ser	Ser	Cys	Cys	Arg	Pro	Ser	Cys	Cys	Val	Ser	Arg
		115					120					125			
Cys	Cys	Arg	Pro	Gln	Cys	Cys	Gln	Ser	Val	Cys	Cys	Gln	Pro	Thr	Cys
		130				135					140				
Cys	Arg	Pro	Ser	Cys	Cys	Ile	Ser	Ser	Cys	Cys	Arg	Pro	Ser	Cys	Cys
145					150					155					160
Glu	Ser	Ser	Cys	Cys	Arg	Pro	Cys	Cys	Cys	Arg	Pro	Cys	Cys	Cys	Leu
			165						170					175	
Arg	Pro	Val	Cys	Gly	Arg	Val	Ser	Cys	His	Thr	Thr	Cys	Tyr	Arg	Pro
			180					185					190		
Thr	Cys	Val	Ile	Ser	Thr	Cys	Pro	Arg	Pro	Leu	Cys	Cys	Ala	Ser	Ser
		195					200					205			

Cys Cys

210

<210> 410

<211> 195

<212> PRT

<213> Homo sapiens

<400> 410
 Met Val Asn Ser Cys Cys Gly Ser Val Cys Ser His Gln Gly Cys Gly
 1 5 10 15
 Gln Asp Leu Cys Gln Glu Thr Cys Cys Arg Pro Ser Cys Cys Glu Thr
 20 25 30
 Thr Cys Cys Arg Thr Thr Tyr Cys Arg Pro Ser Cys Cys Val Ser Ser
 35 40 45
 Cys Cys Arg Pro Gln Cys Cys Gln Ser Val Cys Cys Gln Pro Thr Cys
 50 55 60
 Cys Arg Pro Arg Cys Cys Ile Ser Ser Cys Cys Arg Pro Ser Cys Cys
 65 70 75 80
 Val Ser Ser Cys Cys Lys Pro Gln Cys Cys Gln Ser Met Cys Cys Gln
 85 90 95
 Pro Thr Cys Cys Arg Pro Arg Cys Cys Ile Ser Ser Cys Cys Arg Pro
 100 105 110
 Ser Cys Cys Val Ser Ser Cys Cys Arg Pro Gln Cys Cys Gln Ser Val
 115 120 125
 Cys Cys Gln Pro Thr Cys Cys His Pro Ser Cys Ser Ile Ser Ser Cys
 130 135 140
 Cys Arg Pro Ser Cys Cys Glu Ser Ser Cys Cys Arg Pro Cys Cys Cys
 145 150 155 160
 Leu Arg Pro Val Cys Gly Gly Val Ser Cys His Thr Thr Cys Tyr Arg
 165 170 175
 Pro Thr Cys Val Ile Ser Ser Cys Pro Arg Pro Leu Cys Cys Ala Ser
 180 185 190
 Ser Cys Cys
 195

<210> 411

<211> 201

<212> PRT

<213> Homo sapiens

<400> 411
 Met Val Asn Ser Cys Cys Gly Ser Val Cys Ser Asp Gln Gly Cys Gly
 1 5 10 15
 Leu Glu Asn Cys Cys Arg Pro Ser Cys Cys Gln Thr Thr Cys Cys Arg
 20 25 30
 Thr Thr Cys Cys Arg Pro Ser Cys Cys Val Ser Ser Cys Cys Arg Pro
 35 40 45
 Gln Cys Cys Gln Ser Val Cys Cys Gln Pro Thr Cys Cys Arg Pro Ser
 50 55 60
 Cys Cys Gln Thr Thr Cys Cys Arg Thr Thr Cys Cys Arg Pro Ser Cys
 65 70 75 80
 Cys Val Ser Ser Cys Cys Arg Pro Gln Cys Cys Gln Ser Val Cys Cys
 85 90 95
 Gln Pro Thr Cys Cys Arg Pro Ser Cys Cys Gln Thr Thr Cys Cys Arg
 100 105 110
 Thr Thr Cys Cys Arg Pro Ser Cys Cys Val Ser Ser Cys Cys Arg Pro
 115 120 125
 Gln Cys Cys Gln Ser Val Cys Cys Gln Pro Thr Cys Cys Arg Pro Ser
 130 135 140
 Cys Cys Ile Ser Ser Ser Cys Cys Pro Ser Cys Cys Glu Ser Ser Cys
 145 150 155 160
 Cys Arg Pro Cys Cys Leu Arg Pro Val Cys Gly Arg Val Ser Cys
 165 170 175
 His Thr Thr Cys Tyr Arg Pro Thr Cys Val Ile Ser Thr Cys Pro Arg
 180 185 190
 Pro Leu Cys Cys Ala Ser Ser Cys Cys
 195 200

<210> 412

<211> 186

<212> PRT

<213> Homo sapiens

<400> 412

```

Met Val Ser Ser Cys Cys Gly Ser Val Ser Ser Glu Gln Ser Cys Gly
1      5      10      15
Leu Glu Asn Cys Cys Arg Pro Ser Cys Cys Gln Thr Thr Cys Cys Arg
20      25      30
Thr Thr Cys Cys Arg Pro Ser Cys Cys Lys Pro Gln Cys Cys Gln Ser
35      40      45
Val Cys Tyr Gln Pro Thr Cys Cys His Pro Ser Cys Cys Ile Ser Ser
50      55      60
Cys Cys His Pro Tyr Cys Cys Glu Ser Ser Cys Cys Arg Pro Cys Cys
65      70      75      80
Cys Arg Pro Ser Cys Cys Gln Thr Thr Cys Cys Arg Thr Thr Cys Cys
85      90      95
Arg Thr Thr Cys Cys Cys Pro Ser Cys Cys Val Ser Ser Cys Cys Arg
100     105     110
Pro Gln Cys Cys Gln Ser Val Cys Cys Gln Pro Thr Cys Cys Arg Pro
115     120     125
Ser Cys Cys Ile Ser Ser Cys Cys His Pro Ser Cys Cys Glu Ser Ser
130     135     140
Cys Cys Arg Pro Cys Cys Cys Val Arg Pro Val Cys Gly Arg Val Ser
145     150     155     160
Cys His Thr Thr Cys Tyr Arg Pro Thr Cys Val Ile Ser Thr Cys Pro
165     170     175
Arg Pro Leu Cys Cys Ala Ser Ser Cys Cys
180     185

```

<210> 413

<211> 106

<212> PRT

<213> Homo sapiens

<400> 413

```

Met Val Asn Ser Cys Cys Gly Ser Val Cys Ser Asp Gln Gly Cys Gly
1      5      10      15
Leu Glu Asn Cys Cys Arg Pro Ser Tyr Cys Gln Thr Thr Cys Cys Arg
20      25      30
Thr Thr Cys Cys Arg Pro Ser Cys Cys Arg Pro Ser Cys Cys Arg Pro
35      40      45
Gln Cys Cys Gln Ser Val Cys Cys Gln Pro Thr Cys Cys Cys Pro Ser
50      55      60
Tyr Cys Val Ser Ser Cys Cys Arg Pro Gln Cys Cys Gln Thr Thr Arg
65      70      75      80
Cys Arg Thr Thr Cys Cys Arg Pro Ser Cys Cys Val Ser Arg Cys Tyr
85      90      95
Arg Pro His Cys Gly Gln Ser Leu Cys Cys
100     105

```

<210> 414

<211> 166

<212> PRT

<213> Homo sapiens

<400> 414

Met	Val	Asn	Ser	Cys	Cys	Gly	Ser	Val	Cys	Ser	Asp	Gln	Gly	Cys	Gly
1				5					10					15	
Leu	Glu	Asn	Cys	Cys	Arg	Pro	Ser	Tyr	Cys	Gln	Thr	Thr	Cys	Cys	Arg
			20					25					30		
Thr	Thr	Cys	Cys	Arg	Pro	Ser	Cys	Cys	Val	Ser	Ser	Cys	Cys	Arg	Pro
		35					40					45			
Gln	Cys	Cys	Gln	Thr	Thr	Cys	Cys	Arg	Thr	Thr	Cys	Cys	His	Pro	Ser
	50					55					60				
Cys	Cys	Val	Ser	Ser	Cys	Cys	Arg	Pro	Gln	Cys	Cys	Gln	Ser	Val	Cys
65					70					75					80
Cys	Gln	Pro	Thr	Cys	Cys	Arg	Pro	Gln	Cys	Cys	Gln	Thr	Thr	Cys	Cys
			85						90					95	
Arg	Thr	Thr	Cys	Cys	Arg	Pro	Ser	Cys	Cys	Arg	Pro	Gln	Cys	Cys	Gln
			100					105					110		
Ser	Val	Cys	Cys	Gln	Pro	Thr	Cys	Cys	Cys	Pro	Ser	Tyr	Cys	Val	Ser
		115					120					125			
Ser	Cys	Cys	Arg	Pro	Gln	Cys	Cys	Gln	Thr	Thr	Cys	Cys	Arg	Thr	Thr
	130					135					140				
Cys	Cys	Arg	Pro	Ser	Cys	Cys	Val	Ser	Arg	Cys	Tyr	Arg	Pro	His	Cys
145					150					155					160
Gly	Gln	Ser	Leu	Cys	Cys										
				165											

<210> 415

<211> 136

<212> PRT

<213> Homo sapiens

<400> 415

Met	Val	Asn	Ser	Cys	Cys	Gly	Ser	Val	Cys	Ser	Asp	Gln	Gly	Cys	Gly
1				5					10					15	
Leu	Glu	Asn	Cys	Cys	Arg	Pro	Ser	Cys	Cys	Gln	Thr	Thr	Cys	Cys	Arg
			20					25					30		
Thr	Thr	Cys	Cys	Arg	Pro	Ser	Cys	Cys	Val	Ser	Ser	Cys	Cys	Arg	Pro
		35					40					45			
Gln	Cys	Cys	Gln	Ser	Val	Cys	Cys	Gln	Pro	Thr	Cys	Cys	Ser	Pro	Ser
	50					55					60				
Cys	Cys	Gln	Thr	Thr	Cys	Cys	Arg	Thr	Thr	Cys	Cys	Arg	Pro	Ser	Cys
65					70					75					80
Cys	Val	Ser	Ser	Cys	Phe	Arg	Pro	Gln	Cys	Cys	Gln	Ser	Val	Cys	Cys
			85						90					95	
Gln	Pro	Thr	Cys	Cys	Arg	Pro	Ser	Cys	Gly	Gln	Thr	Thr	Cys	Cys	Arg
			100					105					110		
Thr	Thr	Cys	Tyr	Arg	Pro	Ser	Cys	Cys	Val	Ser	Thr	Cys	Cys	Arg	Pro
		115					120					125			
Thr	Cys	Ser	Ser	Gly	Ser	Cys	Cys								
	130					135									

<210> 416

<211> 127

<212> PRT

<213> Homo sapiens

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<400> 416

Met Val Asn Ser Cys Cys Gly Ser Val Cys Ser Asp Gln Gly Cys Asp
 1 5 10 15
 Gln Gly Leu Cys Gln Glu Thr Cys Cys Arg Pro Ser Cys Cys Gln Thr
 20 25 30
 Thr Cys Cys Cys Pro Ser Cys Val Val Ser Ser Cys Cys Arg Pro Ser
 35 40 45
 Cys Ser Gln Thr Thr Cys Cys Gln Thr Thr Cys Cys Arg Pro Ser Cys
 50 55 60
 Cys Arg Pro Val Cys Cys Gln Thr Thr Cys Arg Pro Ser Cys Gly Val
 65 70 75 80
 Ser Ser Cys Cys Arg Pro Leu Cys Cys Gln Thr Thr Cys Arg Pro Ser
 85 90 95
 Cys Gly Val Ser Ser Cys Cys Arg Pro Leu Cys Cys Gln Thr Thr Cys
 100 105 110
 Cys Arg Thr Thr Cys Cys Arg Pro Ser Cys Cys Gly Ser Ser Cys
 115 120 125

<210> 417

<211> 174

<212> PRT

<213> Homo sapiens

<400> 417

Met Thr His Cys Cys Ser Pro Cys Cys Gln Pro Thr Cys Cys Arg Thr
 1 5 10 15
 Thr Cys Cys Arg Thr Thr Cys Trp Lys Pro Thr Thr Val Thr Thr Cys
 20 25 30
 Ser Ser Thr Pro Cys Cys Gln Pro Ala Cys Cys Val Ser Ser Cys Cys
 35 40 45
 Gln Pro Cys Cys Arg Pro Thr Cys Cys Gln Asn Thr Cys Cys Arg Thr
 50 55 60
 Thr Cys Cys Gln Pro Thr Cys Val Thr Ser Cys Cys Gln Pro Ser Cys
 65 70 75 80
 Cys Ser Thr Pro Cys Cys Gln Pro Thr Cys Cys Gly Ser Ser Cys Cys
 85 90 95
 Gly Gln Thr Ser Cys Gly Ser Ser Cys Gly Gln Ser Ser Ser Cys Ala
 100 105 110
 Pro Val Tyr Cys Arg Arg Thr Cys Tyr Tyr Pro Thr Thr Val Cys Leu
 115 120 125
 Pro Gly Cys Leu Asn Gln Ser Cys Gly Ser Asn Cys Cys Gln Pro Cys
 130 135 140
 Cys Arg Pro Ala Cys Cys Glu Thr Thr Cys Cys Arg Thr Thr Cys Phe
 145 150 155 160
 Gln Pro Thr Cys Val Ser Ser Cys Cys Gln Pro Ser Cys Cys
 165 170

<210> 418

<211> 159

<212> PRT

<213> Homo sapiens

<400> 418

Met Thr His Cys Cys Ser Pro Cys Cys Gln Pro Thr Cys Cys Arg Thr
 1 5 10 15
 Thr Cys Trp Gln Pro Thr Thr Val Thr Thr Cys Ser Ser Thr Pro Cys
 20 25 30

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Cys Gln Pro Ser Cys Cys Val Ser Ser Cys Cys Gln Pro Cys Cys His
 35 40 45
 Pro Thr Cys Cys Gln Asn Thr Cys Cys Arg Thr Thr Cys Cys Gln Pro
 50 55 60
 Ile Cys Val Thr Ser Cys Cys Gln Pro Ser Cys Cys Ser Thr Pro Cys
 65 70 75 80
 Cys Gln Pro Thr Cys Cys Gly Ser Ser Cys Gly Gln Ser Ser Ser Cys
 85 90 95
 Ala Pro Val Tyr Cys Arg Arg Thr Cys Tyr His Pro Thr Ser Val Cys
 100 105 110
 Leu Pro Gly Cys Leu Asn Gln Ser Cys Gly Ser Asn Cys Cys Gln Pro
 115 120 125
 Cys Cys Arg Pro Ala Cys Cys Glu Thr Thr Cys Cys Arg Thr Thr Cys
 130 135 140
 Phe Gln Pro Thr Cys Val Tyr Ser Cys Cys Gln Pro Ser Cys Cys
 145 150 155
 <210> 419
 <211> 159
 <212> PRT
 <213> Homo sapiens

<400> 419
 Met Thr His Cys Cys Ser Pro Cys Cys Gln Pro Thr Cys Cys Arg Thr
 1 5 10 15
 Thr Cys Trp Lys Pro Thr Thr Val Thr Thr Cys Ser Ser Thr Pro Cys
 20 25 30
 Cys Gln Pro Ser Cys Cys Val Ser Ser Cys Cys Gln Pro Ile Cys Val Thr Ser
 35 40 45
 Pro Thr Cys Cys Gln Asn Thr Cys Cys Gln Pro Ile Cys Val Thr Ser
 50 55 60
 Cys Cys Gln Pro Ser Cys Cys Ser Thr Pro Cys Cys Gln Pro Thr Cys
 65 70 75 80
 Cys Gly Gln Thr Ser Cys Gly Ser Ser Cys Gly Gln Ser Ser Ser Cys
 85 90 95
 Ala Pro Val Tyr Cys Arg Arg Thr Cys Tyr His Pro Thr Thr Val Cys
 100 105 110
 Leu Pro Gly Cys Leu Asn Gln Ser Cys Gly Ser Ser Cys Cys Gln Pro
 115 120 125
 Cys Cys Arg Pro Ala Cys Cys Glu Thr Thr Cys Cys Arg Thr Thr Cys
 130 135 140
 Phe Gln Pro Thr Cys Val Tyr Ser Cys Cys Gln Pro Ser Cys Cys
 145 150 155
 <210> 420
 <211> 154
 <212> PRT
 <213> Homo sapiens

<400> 420
 Met Thr His Cys Cys Ser Pro Cys Cys Gln Pro Thr Cys Cys Arg Thr
 1 5 10 15
 Thr Cys Cys Arg Thr Thr Cys Trp Lys Pro Thr Thr Val Thr Thr Cys
 20 25 30
 Ser Ser Thr Pro Cys Cys Gln Pro Ser Cys Cys Val Ser Ser Cys Cys
 35 40 45
 Gln Pro Cys Cys Arg Pro Ala Cys Cys Gln Asn Thr Cys Cys Arg Thr
 50 55 60

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Thr Cys Cys Gln Pro Thr Cys Leu Ser Ser Cys Cys Gly Gln Thr Ser
65 70 75 80
Cys Gly Ser Ser Cys Gly Gln Ser Ser Ser Cys Ala Pro Val Tyr Cys
85 90 95
Arg Arg Thr Cys Tyr Tyr Pro Thr Thr Val Cys Leu Pro Gly Cys Leu
100 105 110
Asn Gln Ser Cys Gly Ser Ser Cys Cys Gln Pro Cys Cys Arg Pro Ala
115 120 125
Cys Cys Glu Thr Thr Cys Cys Arg Thr Thr Cys Phe Gln Pro Thr Cys
130 135 140
Val Ser Ser Cys Cys Gln Pro Ser Cys Cys
145 150

<210> 421

<211> 154

<212> PRT

<213> Homo sapiens

<400> 421

Met Thr His Cys Cys Ser Pro Cys Cys Gln Pro Thr Cys Cys Arg Thr
1 5 10 15
Thr Cys Cys Arg Thr Thr Cys Trp Lys Pro Thr Thr Val Thr Thr Cys
20 25 30
Ser Ser Thr Pro Cys Cys Gln Pro Ser Cys Cys Val Ser Ser Cys Cys
35 40 45
Gln Pro Cys Cys Arg Pro Thr Cys Cys Gln Asn Thr Cys Cys Gln Pro
50 55 60
Thr Cys Val Thr Ser Cys Cys Gln Pro Ser Cys Cys Ser Thr Pro Cys
65 70 75 80
Cys Gln Pro Thr Cys Cys Gly Ser Ser Cys Asp Gln Ser Ser Ser Cys
85 90 95
Ala Pro Val Tyr Cys Arg Arg Thr Cys Tyr Tyr Pro Thr Thr Val Cys
100 105 110
Leu Pro Gly Cys Leu Asn Gln Ser Cys Gly Ser Asn Cys Cys Gln Pro
115 120 125
Cys Cys Arg Pro Ala Cys Cys Glu Thr Thr Cys Phe Gln Pro Thr Cys
130 135 140
Val Ser Ser Cys Cys Gln Pro Phe Cys Cys
145 150

<210> 422

<211> 138

<212> PRT

<213> Homo sapiens

<400> 422

Met Leu Gln Asp His Leu Leu Gln Asp Asn Leu Leu Glu Ala His His
1 5 10 15
Cys Asp His Leu Gln Gln His Ile Leu Leu Pro Ala Leu Leu Leu Cys
20 25 30
Val Gln Leu Leu Pro Ala Leu Leu Pro Pro Asn Leu Leu Ser Lys His
35 40 45
Leu Leu Gln Asp His Leu Leu Pro Ala His Leu Cys Asp Gln Leu Leu
50 55 60
Pro Ala Phe Leu Leu Gln His Thr Leu Leu Thr Ala His Leu Leu Trp
65 70 75 80
Val Gln Leu Leu Trp Pro Asn His Leu Trp Val Gln Leu Leu Pro Ala
85 90 95

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Gln Leu Leu Cys Thr His Leu Leu Gln Glu Asn Leu Leu Pro Pro His
 100 105 110
 Glu Cys Leu Pro Ala Trp Leu Pro Lys Ser Glu Leu Trp Leu Gln Leu
 115 120 125
 Leu Pro Ala Leu Leu Pro Pro Ser Leu Leu
 130 135

<210> 423

<211> 409

<212> PRT

<213> Homo sapiens

<400> 423

Met Ser Gly Ser Cys Ser Ser Arg Lys Cys Phe Ser Val Pro Ala Thr
 1 5 10 15
 Ser Leu Cys Ser Thr Glu Val Ser Cys Gly Gly Pro Ile Cys Leu Pro
 20 25 30
 Ser Ser Cys Gln Ser Gln Thr Trp Gln Leu Val Thr Cys Gln Asp Ser
 35 40 45
 Cys Gly Ser Ser Ser Cys Gly Pro Gln Cys Arg Gln Pro Ser Cys Pro
 50 55 60
 Val Ser Ser Cys Ala Gln Pro Leu Cys Cys Asp Pro Val Ile Cys Glu
 65 70 75 80
 Pro Ser Cys Ser Val Ser Ser Gly Cys Gln Pro Val Cys Cys Glu Ala
 85 90 95
 Thr Thr Cys Glu Pro Ser Cys Ser Val Ser Asn Cys Tyr Gln Pro Val
 100 105 110
 Cys Phe Glu Ala Thr Ile Cys Glu Pro Ser Cys Ser Val Ser Asn Cys
 115 120 125
 Cys Gln Pro Val Cys Phe Glu Ala Thr Val Cys Glu Pro Ser Cys Ser
 130 135 140
 Val Ser Ser Cys Ala Gln Pro Val Cys Cys Glu Pro Ala Ile Cys Glu
 145 150 155 160
 Pro Ser Cys Ser Val Ser Ser Cys Cys Gln Pro Val Gly Ser Glu Ala
 165 170 175
 Thr Ser Cys Gln Pro Val Leu Cys Val Pro Thr Ser Cys Gln Pro Val
 180 185 190
 Leu Cys Lys Ser Ser Cys Cys Gln Pro Val Val Cys Glu Pro Ser Cys
 195 200 205
 Cys Ser Ala Val Cys Thr Leu Pro Ser Ser Cys Gln Pro Val Val Cys
 210 215 220
 Glu Pro Ser Cys Cys Gln Pro Val Cys Pro Thr Pro Thr Cys Ser Val
 225 230 235 240
 Thr Ser Ser Cys Gln Ala Val Cys Cys Asp Pro Ser Pro Trp Ser Ser
 245 250 255
 Ala Ser Ala Ile Cys Arg Pro Thr Cys Pro Arg Thr Phe Tyr Ile Pro
 260 265 270
 Ser Ser Ser Lys Arg Pro Cys Ser Ala Thr Ile Ser Tyr Arg Pro Val
 275 280 285
 Ser Arg Pro Ile Cys Arg Pro Ile Cys Ser Gly Leu Leu Thr Tyr Arg
 290 295 300
 Gln Pro Tyr Met Thr Ser Ile Ser Tyr Arg Pro Ala Cys Tyr Arg Pro
 305 310 315 320
 Cys Tyr Ser Ile Leu Arg Arg Pro Ala Cys Val Thr Ser Tyr Ser Cys
 325 330 335
 Arg Pro Val Tyr Phe Arg Pro Ser Cys Thr Glu Ser Asp Ser Cys Lys
 340 345 350
 Arg Asp Cys Lys Lys Ser Thr Ser Ser Gln Leu Asp Cys Val Asp Thr
 355 360 365
 Thr Pro Cys Lys Val Asp Val Ser Glu Glu Ala Pro Cys Gln Pro Thr
 370 375 380

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Glu Ala Lys Pro Ile Ser Pro Thr Thr Arg Glu Ala Ala Ala Ala Gln
 385 390 395 400
 Pro Ala Ala Ser Lys Pro Ala Asn Cys
 405

<210> 424

<211> 105

<212> PRT

<213> Homo sapiens

<400> 424

Met Gly Cys Cys Pro Gly Asp Cys Phe Thr Cys Cys Thr Gln Glu Gln
 1 5 10 15
 Asn Cys Cys Glu Glu Cys Cys Cys Gln Pro Gly Cys Cys Gly Cys Cys
 20 25 30
 Gly Ser Cys Cys Gly Cys Gly Gly Ser Gly Cys Gly Gly Ser Gly Cys
 35 40 45
 Gly Gly Ser Cys Cys Gly Ser Ser Cys Cys Gly Ser Gly Cys Gly Gly
 50 55 60
 Cys Gly Gly Cys Gly Gly Cys Gly Gly Gly Cys Cys Gly Ser Ser Cys
 65 70 75 80
 Cys Gly Ser Ser Cys Cys Gly Ser Gly Cys Cys Gly Pro Val Cys Cys
 85 90 95
 Gln Pro Thr Pro Ile Cys Asp Thr Lys
 100 105

<210> 425

<211> 404

<212> PRT

<213> Homo sapiens

<400> 425

Met Ser Tyr Ser Cys Gly Leu Pro Ser Leu Ser Cys Arg Thr Ser Cys
 1 5 10 15
 Ser Ser Arg Pro Cys Val Pro Pro Ser Cys His Gly Cys Thr Leu Pro
 20 25 30
 Gly Ala Cys Asn Ile Pro Ala Asn Val Ser Asn Cys Asn Trp Phe Cys
 35 40 45
 Glu Gly Ser Phe Asn Gly Ser Glu Lys Glu Thr Met Gln Phe Leu Asn
 50 55 60
 Asp Arg Leu Ala Ser Tyr Leu Glu Lys Val Arg Gln Leu Glu Arg Asp
 65 70 75 80
 Asn Ala Glu Leu Glu Asn Leu Ile Arg Glu Arg Ser Gln Gln Gln Glu
 85 90 95
 Pro Leu Val Cys Ala Ser Tyr Gln Ser Tyr Phe Lys Thr Ile Glu Glu
 100 105 110
 Leu Gln Gln Lys Ile Leu Cys Ser Lys Ser Glu Asn Ala Arg Leu Val
 115 120 125
 Val Gln Ile Asp Asn Ala Lys Leu Ala Ser Asp Asp Phe Arg Thr Lys
 130 135 140
 Tyr Glu Thr Glu Leu Ser Leu Arg Gln Leu Val Glu Ser Asp Ile Asn
 145 150 155 160
 Gly Leu Arg Arg Ile Leu Asp Glu Leu Thr Leu Cys Arg Ser Asp Leu
 165 170 175
 Glu Ala Gln Val Glu Ser Leu Lys Glu Glu Leu Leu Cys Leu Lys Gln
 180 185 190
 Asn His Glu Gln Glu Val Asn Thr Leu Arg Cys Gln Leu Gly Asp Arg
 195 200 205

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Leu Asn Val Glu Val Asp Ala Ala Pro Thr Val Asp Leu Asn Gln Val
 210      215      220
Leu Asn Glu Thr Arg Ser Gln Tyr Glu Ala Leu Val Glu Thr Asn Arg
225      230      235      240
Arg Glu Val Glu Gln Trp Phe Ala Thr Gln Thr Glu Glu Leu Asn Lys
      245      250      255
Gln Val Val Ser Ser Ser Glu Gln Leu Gln Ser Tyr Gln Ala Glu Ile
 260      265      270
Ile Glu Leu Arg Arg Thr Val Asn Ala Leu Glu Ile Glu Leu Gln Ala
 275      280      285
Gln His Asn Leu Arg Asp Ser Leu Glu Asn Thr Leu Thr Glu Ser Glu
 290      295      300
Ala Arg Tyr Ser Ser Gln Leu Ser Gln Val Gln Arg Leu Ile Thr Asn
305      310      315      320
Val Glu Ser Gln Leu Ala Glu Ile Arg Ser Asp Leu Glu Arg Gln Asn
      325      330      335
Gln Glu Tyr Gln Val Leu Leu Asp Val Arg Ala Arg Leu Glu Cys Glu
 340      345      350
Ile Asn Thr Tyr Arg Ser Leu Leu Glu Ser Glu Asp Cys Lys Leu Pro
 355      360      365
Ser Asn Pro Cys Ala Thr Thr Asn Ala Cys Asp Lys Ser Thr Gly Pro
 370      375      380
Cys Ile Ser Asn Pro Cys Gly Leu Arg Ala Arg Cys Gly Pro Cys Asn
385      390      395      400
Thr Phe Gly Tyr

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<210> 426

<211> 404

<212> PRT

<213> Homo sapiens

<400> 426

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Met Pro Tyr Asn Phe Cys Leu Pro Ser Leu Ser Cys Arg Thr Ser Cys
 1      5      10      15
Ser Ser Arg Pro Cys Val Pro Pro Ser Cys His Gly Tyr Thr Leu Pro
      20      25      30
Gly Ala Cys Asn Ile Pro Ala Asn Val Ser Asn Cys Asn Trp Phe Cys
 35      40      45
Glu Gly Ser Phe Asn Gly Ser Glu Lys Glu Thr Met Gln Phe Leu Asn
 50      55      60
Asp Arg Leu Ala Ser Tyr Leu Glu Lys Val Arg Gln Leu Glu Arg Asp
 65      70      75      80
Asn Ala Glu Leu Glu Asn Leu Ile Arg Glu Arg Ser Gln Gln Gln Glu
      85      90      95
Pro Leu Leu Cys Pro Ser Tyr Gln Ser Tyr Phe Lys Thr Ile Glu Glu
 100      105      110
Leu Gln Gln Lys Ile Leu Cys Ser Lys Ser Glu Asn Ala Arg Leu Val
 115      120      125
Val Gln Ile Asp Asn Ala Lys Leu Ala Ala Asp Asp Phe Arg Thr Lys
 130      135      140
Tyr Gln Thr Glu Gln Ser Leu Arg Gln Leu Val Glu Ser Asp Ile Asn
 145      150      155      160
Ser Leu Arg Arg Ile Leu Asp Glu Leu Thr Leu Cys Arg Ser Asp Leu
      165      170      175
Glu Ala Gln Met Glu Ser Leu Lys Glu Leu Leu Ser Leu Lys Gln
 180      185      190
Asn His Glu Gln Glu Val Asn Thr Leu Arg Cys Gln Leu Gly Asp Arg
 195      200      205
Leu Asn Val Glu Val Asp Ala Ala Pro Ala Val Asp Leu Asn Gln Val
 210      215      220

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Leu Asn Glu Thr Arg Asn Gln Tyr Glu Ala Leu Val Glu Thr Asn Arg
 225 230 235 240
 Arg Glu Val Glu Gln Trp Phe Ala Thr Gln Thr Glu Glu Leu Asn Lys
 245 250 255
 Gln Val Val Ser Ser Ser Glu Gln Leu Gln Ser Tyr Gln Ala Glu Ile
 260 265 270
 Ile Glu Leu Arg Arg Thr Val Asn Ala Leu Glu Ile Glu Leu Gln Ala
 275 280 285
 Gln His Asn Leu Arg Tyr Ser Leu Glu Asn Thr Leu Thr Glu Ser Glu
 290 295 300
 Ala Arg Tyr Ser Ser Gln Leu Ser Gln Val Gln Ser Leu Ile Thr Asn
 305 310 315 320
 Val Glu Ser Gln Leu Ala Glu Ile Arg Ser Asp Leu Glu Arg Gln Asn
 325 330 335
 Gln Glu Tyr Gln Val Leu Leu Asp Val Arg Ala Arg Leu Glu Cys Glu
 340 345 350
 Ile Asn Thr Tyr Arg Ser Leu Leu Glu Ser Glu Asp Cys Lys Leu Pro
 355 360 365
 Ser Asn Pro Cys Ala Thr Thr Asn Ala Cys Glu Lys Pro Ile Gly Ser
 370 375 380
 Cys Val Thr Asn Pro Cys Gly Pro Arg Ser Arg Cys Gly Pro Cys Asn
 385 390 395 400
 Thr Phe Gly Tyr

<210> 427

<211> 436

<212> PRT

<213> Homo sapiens

<400> 427

Met Leu Tyr Ala Lys Pro Pro Pro Thr Ile Asn Gly Ile Lys Gly Leu
 1 5 10 15
 Gln Arg Lys Glu Arg Leu Lys Pro Ala His Ile His Leu Gln Gln Leu
 20 25 30
 Thr Cys Phe Ser Ile Thr Cys Ser Ser Thr Met Ser Tyr Ser Cys Cys
 35 40 45
 Leu Pro Ser Leu Gly Cys Arg Thr Ser Cys Ser Ser Arg Pro Cys Val
 50 55 60
 Pro Pro Ser Cys His Gly Tyr Thr Leu Pro Gly Ala Cys Asn Ile Pro
 65 70 75 80
 Ala Asn Val Ser Asn Cys Asn Trp Phe Cys Glu Gly Ser Phe Asn Gly
 85 90 95
 Ser Glu Lys Glu Thr Met Gln Phe Leu Asn Asp Arg Leu Ala Ser Tyr
 100 105 110
 Leu Glu Lys Val Arg Gln Leu Glu Arg Asp Asn Ala Glu Leu Glu Lys
 115 120 125
 Leu Ile Gln Glu Arg Ser Gln Gln Gln Glu Pro Leu Leu Cys Pro Ser
 130 135 140
 Tyr Gln Ser Tyr Phe Lys Thr Ile Glu Glu Leu Gln Gln Lys Ile Leu
 145 150 155 160
 Cys Ala Lys Ala Glu Asn Ala Arg Leu Val Val Asn Ile Asp Asn Ala
 165 170 175
 Lys Leu Ala Ser Asp Asp Phe Arg Ser Lys Tyr Gln Thr Glu Gln Ser
 180 185 190
 Leu Arg Leu Leu Val Glu Ser Asp Ile Asn Ser Ile Arg Arg Ile Leu
 195 200 205
 Asp Glu Leu Thr Leu Cys Lys Ser Asp Leu Glu Ser Gln Val Glu Ser
 210 215 220
 Leu Arg Glu Glu Leu Ile Cys Leu Lys Lys Asn His Glu Glu Glu Val
 225 230 235 240

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Asn Thr Leu Arg Ser Gln Leu Gly Asp Arg Leu Asn Val Glu Val Asp
 245 250 255
 Thr Ala Pro Thr Val Asp Leu Asn Gln Val Leu Asn Glu Thr Arg Ser
 260 265 270
 Gln Tyr Glu Ala Leu Val Glu Ile Asn Arg Arg Glu Val Glu Gln Trp
 275 280 285
 Phe Ala Thr Gln Thr Glu Glu Leu Asn Lys Gln Val Val Ser Ser Ser
 290 295 300
 Glu Gln Leu Gln Ser Cys Gln Ala Glu Ile Ile Glu Leu Arg Arg Thr
 305 310 315 320
 Val Asn Ala Leu Glu Ile Glu Leu Gln Ala Gln His Asn Leu Arg Asp
 325 330 335
 Ser Leu Glu Asn Thr Leu Thr Glu Ser Glu Ala His Tyr Ser Ser Gln
 340 345 350
 Leu Ser Gln Val Gln Ser Leu Ile Thr Asn Val Glu Ser Gln Leu Ala
 355 360 365
 Glu Ile Arg Cys Asp Leu Glu Arg Gln Asn Gln Glu Tyr Gln Val Leu
 370 375 380
 Leu Asp Val Arg Ala Arg Leu Glu Cys Glu Ile Asn Thr Tyr Arg Ser
 385 390 395 400
 Leu Leu Glu Ser Glu Asp Cys Lys Leu Pro Cys Asn Pro Cys Ala Thr
 405 410 415
 Thr Asn Ala Ser Gly Asn Ser Cys Gly Pro Cys Gly Thr Ser Gln Lys
 420 425 430
 Gly Cys Cys Asn
 435

<210> 428

<211> 416

<212> PRT

<213> Homo sapiens

<400> 428

Met Pro Tyr Asn Phe Cys Leu Pro Ser Leu Ser Cys Arg Thr Ser Cys
 1 5 10 15
 Ser Ser Arg Pro Cys Val Pro Pro Ser Cys His Ser Cys Thr Leu Pro
 20 25 30
 Gly Ala Cys Asn Ile Pro Ala Asn Val Ser Asn Cys Asn Trp Phe Cys
 35 40 45
 Glu Gly Ser Phe Asn Gly Ser Glu Lys Glu Thr Met Gln Phe Leu Asn
 50 55 60
 Asp Arg Leu Ala Ser Tyr Leu Glu Lys Val Arg Gln Leu Glu Arg Asp
 65 70 75 80
 Asn Ala Glu Leu Glu Asn Leu Ile Arg Glu Arg Ser Gln Gln Gln Glu
 85 90 95
 Pro Leu Leu Cys Pro Ser Tyr Gln Ser Tyr Phe Lys Thr Ile Glu Glu
 100 105 110
 Leu Gln Gln Lys Ile Leu Cys Thr Lys Ser Glu Asn Ala Arg Leu Val
 115 120 125
 Val Gln Ile Asp Asn Ala Lys Leu Ala Ala Asp Asp Phe Arg Thr Lys
 130 135 140
 Tyr Gln Thr Glu Leu Ser Leu Arg Gln Leu Val Glu Ser Asp Ile Asn
 145 150 155 160
 Gly Leu Arg Arg Ile Leu Asp Glu Leu Thr Leu Cys Lys Ser Asp Leu
 165 170 175
 Glu Ala Gln Val Glu Ser Leu Lys Glu Glu Leu Cys Leu Lys Ser
 180 185 190
 Asn His Glu Gln Glu Val Asn Thr Leu Arg Cys Gln Leu Gly Asp Arg
 195 200 205
 Leu Asn Val Glu Val Asp Ala Ala Pro Thr Val Asp Leu Asn Arg Val
 210 215 220

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Leu Asn Glu Thr Arg Ser Gln Tyr Glu Ala Leu Val Glu Thr Asn Arg
 225 230 235 240
 Arg Glu Val Glu Gln Trp Phe Thr Thr Gln Thr Glu Glu Leu Asn Lys
 245 250 255
 Gln Val Val Ser Ser Ser Glu Gln Leu Gln Ser Tyr Gln Ala Glu Ile
 260 265 270
 Ile Glu Leu Arg Arg Thr Val Asn Ala Leu Glu Ile Glu Leu Gln Ala
 275 280 285
 Gln His Asn Leu Arg Asp Ser Leu Glu Asn Thr Leu Thr Glu Ser Glu
 290 295 300
 Ala Arg Tyr Ser Ser Gln Leu Ser Gln Val Gln Ser Leu Ile Thr Asn
 305 310 315 320
 Val Glu Ser Gln Leu Ala Glu Ile Arg Ser Asp Leu Glu Arg Gln Asn
 325 330 335
 Gln Glu Tyr Gln Val Leu Leu Asp Val Arg Ala Arg Leu Glu Cys Glu
 340 345 350
 Ile Asn Thr Tyr Arg Ser Leu Leu Glu Ser Glu Asp Cys Asn Leu Pro
 355 360 365
 Ser Asn Pro Cys Ala Thr Thr Asn Ala Cys Ser Lys Pro Ile Gly Pro
 370 375 380
 Cys Leu Ser Asn Pro Cys Thr Ser Cys Val Pro Pro Ala Pro Cys Thr
 385 390 395 400
 Pro Cys Ala Pro Arg Pro Arg Cys Gly Pro Cys Asn Ser Phe Val Arg
 405 410 415

<210> 429

<211> 201

<212> PRT

<213> Homo sapiens

<400> 429

Met Thr Ser Asp His Cys Ser Ser Leu Leu Ser Gly Gln Val Ser Glu
 1 5 10 15
 Ala Asn Ala Ala Ser Leu Cys Leu Leu Ala Asn Val Ala His Ala Asn
 20 25 30
 Arg Val Arg Val Gly Ser Thr Pro Leu Gly Arg Leu Ser Leu Cys Leu
 35 40 45
 Pro Pro Thr Cys His Thr Thr Cys Pro Leu Pro Gly Thr Cys His Ile
 50 55 60
 Pro Gly Asn Ile Gly Ile Cys Gly Ala Tyr Arg Glu Asn Thr Leu Asn
 65 70 75 80
 Gly His Glu Lys Glu Thr Met Gln Phe Leu Asn Asp Arg Leu Ala Asn
 85 90 95
 Tyr Leu Glu Lys Val Arg Gln Leu Glu Trp Asp Asn Ala Glu Leu Glu
 100 105 110
 Thr Lys Leu His Glu Arg Ser Lys Cys His Glu Ser Ser Val Cys Arg
 115 120 125
 Asn Tyr Gln Ser Tyr Phe Cys Thr Ile Gln Glu Leu Gln Gln Lys Val
 130 135 140
 Arg Phe Ala Val His Gln Ile Arg Gly Gln Glu Ser Ala Tyr Cys Leu
 145 150 155 160
 Ser Ala Lys Ser Gly Pro Pro Pro Ala Phe Ala Asn Lys Val Leu Leu
 165 170 175
 Val His Gly His Ala His Ala Phe Val Cys Cys Leu Gln Leu Leu Leu
 180 185 190
 Tyr Tyr Ser Gly Arg Val Gln Ser Leu
 195 200

<210> 430

<211> 471

<212> PRT

<210> 431

<211> 456

<212> PRT

<213> Homo sapiens

<400> 431

Met	Thr	Ser	Ser	Tyr	Ser	Ser	Ser	Ser	Ser	Cys	Pro	Leu	Gly	Cys	Thr	Met
1				5						10					15	
Ala	Pro	Gly	Ala	Arg	Asn	Val	Ser	Val	Ser	Pro	Ile	Asp	Ile	Gly	Cys	
			20					25					30			
Gln	Pro	Gly	Ala	Glu	Ala	Asn	Ile	Ala	Pro	Met	Cys	Leu	Leu	Ala	Asn	
		35				40						45				
Val	Ala	His	Ala	Asn	Arg	Val	Arg	Val	Gly	Ser	Thr	Pro	Leu	Gly	Arg	
	50				55						60					
Pro	Ser	Leu	Cys	Leu	Pro	Pro	Thr	Cys	His	Thr	Ala	Cys	Pro	Leu	Pro	
65					70					75					80	
Gly	Thr	Cys	His	Ile	Pro	Gly	Asn	Ile	Gly	Ile	Cys	Gly	Ala	Tyr	Gly	
			85						90					95		
Glu	Asn	Thr	Leu	Asn	Gly	His	Glu	Lys	Glu	Thr	Met	Gln	Phe	Leu	Asn	
			100					105					110			
Asp	Arg	Leu	Ala	Asn	Tyr	Leu	Glu	Lys	Val	Arg	Gln	Leu	Glu	Gln	Glu	
		115					120					125				
Asn	Ala	Glu	Leu	Glu	Ala	Thr	Leu	Leu	Glu	Arg	Ser	Lys	Cys	His	Glu	
	130				135						140					
Ser	Thr	Val	Cys	Pro	Asp	Tyr	Gln	Ser	Tyr	Phe	His	Thr	Ile	Glu	Glu	
145					150					155					160	
Leu	Gln	Gln	Lys	Ile	Leu	Cys	Ser	Lys	Ala	Glu	Asn	Ala	Arg	Leu	Ile	
			165						170					175		
Val	Gln	Ile	Asp	Asn	Ala	Lys	Leu	Ala	Ala	Asp	Asp	Phe	Arg	Ile	Lys	
			180					185					190			
Leu	Glu	Ser	Glu	Arg	Ser	Leu	Arg	Gln	Leu	Val	Glu	Ala	Asp	Lys	Cys	
		195					200					205				
Gly	Thr	Gln	Lys	Leu	Leu	Asp	Asp	Ala	Thr	Leu	Ala	Lys	Ala	Asp	Leu	
	210					215					220					
Glu	Ala	Gln	Gln	Glu	Ser	Leu	Lys	Glu	Glu	Gln	Leu	Ser	Leu	Lys	Ser	
225					230					235					240	
Asn	His	Glu	Gln	Glu	Val	Lys	Ile	Leu	Arg	Ser	Gln	Leu	Gly	Glu	Lys	
			245						250					255		
Leu	Arg	Ile	Glu	Leu	Asp	Ile	Glu	Pro	Thr	Ile	Asp	Leu	Asn	Arg	Val	
			260					265					270			
Leu	Gly	Glu	Met	Arg	Ala	Gln	Tyr	Glu	Ala	Met	Leu	Glu	Thr	Asn	Arg	
	275						280					285				
Gln	Asp	Val	Glu	Gln	Trp	Phe	Gln	Ala	Gln	Ser	Glu	Gly	Ile	Ser	Leu	
	290					295					300					
Gln	Asp	Met	Ser	Cys	Ser	Glu	Glu	Leu	Gln	Cys	Cys	Gln	Ser	Glu	Ile	
305					310					315					320	
Leu	Glu	Leu	Arg	Cys	Thr	Val	Asn	Ala	Leu	Glu	Val	Glu	Arg	Gln	Ala	
			325						330					335		
Gln	His	Thr	Leu	Lys	Asp	Cys	Leu	Gln	Asn	Ser	Leu	Cys	Glu	Ala	Glu	
			340					345					350			
Asp	Arg	Phe	Gly	Thr	Glu	Leu	Ala	Gln	Met	Gln	Ser	Leu	Ile	Ser	Asn	
	355						360						365			
Val	Glu	Glu	Gln	Leu	Ser	Glu	Ile	Arg	Ala	Asp	Leu	Glu	Arg	Gln	Asn	
	370					375					380					
Gln	Glu	Tyr	Gln	Val	Leu	Leu	Asp	Val	Lys	Thr	Arg	Leu	Glu	Asn	Glu	
385					390					395					400	
Ile	Ala	Thr	Tyr	Arg	Asn	Leu	Leu	Glu	Ser	Glu	Asp	Cys	Lys	Leu	Pro	
				405					410					415		
Cys	Asn	Pro	Cys	Ser	Thr	Ser	Pro	Ser	Cys	Val	Thr	Ala	Pro	Cys	Ala	
			420					425					430			

Pro Arg Pro Ser Cys Gly Pro Cys Thr Thr Cys Gly Pro Thr Cys Gly
 435 440 445
 Ala Ser Thr Thr Gly Ser Arg Phe
 450 455
 <210> 432
 <211> 448
 <212> PRT
 <213> Homo sapiens

<400> 432
 Met Thr Ser Ser Cys Cys Val Thr Asn Asn Leu Gln Ala Ser Leu Lys
 1 5 10 15
 Ser Cys Pro Arg Pro Ala Ser Val Cys Ser Ser Gly Val Asn Cys Arg
 20 25 30
 Pro Glu Leu Cys Leu Gly Tyr Val Cys Gln Pro Met Ala Cys Leu Pro
 35 40 45
 Ser Val Cys Leu Pro Thr Thr Phe Arg Pro Ala Ser Cys Leu Ser Lys
 50 55 60
 Thr Tyr Leu Ser Ser Ser Cys Gln Ala Ala Ser Gly Ile Ser Gly Ser
 65 70 75 80
 Met Gly Pro Gly Ser Trp Tyr Ser Glu Gly Ala Phe Asn Gly Asn Glu
 85 90 95
 Lys Glu Thr Met Gln Phe Leu Asn Asp Arg Leu Ala Ser Tyr Leu Thr
 100 105 110
 Arg Val Arg Gln Leu Glu Gln Glu Asn Ala Glu Leu Glu Ser Arg Ile
 115 120 125
 Gln Glu Ala Ser His Ser Gln Val Leu Thr Met Thr Pro Asp Tyr Gln
 130 135 140
 Ser His Phe Arg Thr Ile Glu Glu Leu Gln Gln Lys Ile Leu Cys Thr
 145 150 155 160
 Lys Ala Glu Asn Ala Arg Met Val Val Asn Ile Asp Asn Ala Lys Leu
 165 170 175
 Ala Ala Asp Asp Phe Arg Ala Lys Tyr Glu Ala Glu Leu Ala Met Arg
 180 185 190
 Gln Leu Val Glu Ala Asp Ile Asn Gly Leu Arg Arg Ile Leu Asp Asp
 195 200 205
 Leu Thr Leu Cys Lys Ala Asp Leu Glu Ala Gln Val Glu Ser Leu Lys
 210 215 220
 Glu Glu Leu Met Cys Leu Lys Lys Asn His Glu Glu Glu Val Gly Ser
 225 230 235 240
 Leu Arg Cys Gln Leu Gly Asp Arg Leu Asn Ile Glu Val Asp Ala Ala
 245 250 255
 Pro Pro Val Asp Leu Thr Arg Val Leu Glu Glu Met Arg Cys Gln Tyr
 260 265 270
 Glu Ala Met Val Glu Ala Asn Arg Arg Asp Val Glu Glu Trp Phe Asn
 275 280 285
 Met Gln Met Glu Glu Leu Asn Gln Gln Val Ala Thr Ser Ser Glu Gln
 290 295 300
 Leu Gln Asn Tyr Gln Ser Asp Ile Ile Asp Leu Arg Arg Thr Val Asn
 305 310 315 320
 Thr Leu Glu Ile Glu Leu Gln Ala Gln His Ser Leu Arg Asp Ser Leu
 325 330 335
 Glu Asn Thr Leu Thr Glu Ser Glu Ala Arg Tyr Ser Ser Gln Leu Ala
 340 345 350
 Gln Met Gln Cys Met Ile Thr Asn Val Glu Ala Gln Leu Ala Glu Ile
 355 360 365
 Arg Ala Asp Leu Glu Arg Gln Asn Gln Glu Tyr Gln Val Leu Leu Asp
 370 375 380
 Val Arg Ala Arg Leu Glu Gly Glu Ile Asn Thr Tyr Arg Ser Leu Leu
 385 390 395 400

Glu Ser Glu Asp Cys Lys Leu Pro Cys Asn Pro Cys Ser Thr Pro Ser
 405 410 415
 Cys Thr Thr Cys Val Pro Ser Pro Cys Val Thr Arg Thr Val Cys Val
 420 425 430
 Pro Arg Thr Val Gly Met Pro Cys Ser Pro Cys Pro Gln Gly Arg Tyr
 435 440 445
 <210> 433
 <211> 425
 <212> PRT
 <213> Homo sapiens

<400> 433
 Met Tyr Ser Ser Ser Ser Cys Lys Leu Pro Ser Leu Ser Pro Val Ala
 1 5 10 15
 Arg Ser Phe Ser Ala Cys Ser Val Gly Leu Gly Arg Ser Ser Tyr Arg
 20 25 30
 Ala Thr Ser Cys Leu Pro Ala Leu Cys Leu Pro Ala Gly Gly Phe Ala
 35 40 45
 Thr Ser Tyr Ser Gly Gly Gly Gly Trp Phe Gly Glu Gly Ile Leu Thr
 50 55 60
 Gly Asn Glu Lys Glu Thr Met Gln Ser Leu Asn Asp Arg Leu Ala Gly
 65 70 75 80
 Tyr Leu Glu Lys Val Arg Gln Leu Glu Gln Glu Asn Ala Ser Leu Glu
 85 90 95
 Ser Arg Ile Arg Glu Trp Cys Glu Gln Gln Val Pro Tyr Met Cys Pro
 100 105 110
 Asp Tyr Gln Ser Tyr Phe Arg Thr Ile Glu Glu Leu Gln Lys Lys Thr
 115 120 125
 Leu Cys Ser Lys Ala Glu Asn Ala Arg Leu Val Val Glu Ile Asp Asn
 130 135 140
 Ala Lys Leu Ala Ala Asp Asp Phe Arg Thr Lys Tyr Glu Thr Glu Val
 145 150 155 160
 Ser Leu Arg Gln Leu Val Glu Ser Asp Ile Asn Gly Leu Arg Arg Ile
 165 170 175
 Leu Asp Asp Leu Thr Leu Cys Lys Ser Asp Leu Glu Ala Gln Val Glu
 180 185 190
 Ser Leu Lys Glu Glu Leu Leu Cys Leu Lys Lys Asn His Glu Glu Glu
 195 200 205
 Val Asn Ser Leu Arg Cys Gln Leu Gly Asp Arg Leu Asn Val Glu Val
 210 215 220
 Asp Ala Ala Pro Pro Val Asp Leu Asn Arg Val Leu Glu Glu Met Arg
 225 230 235 240
 Cys Gln Tyr Glu Thr Leu Val Glu Asn Asn Arg Arg Asp Ala Glu Asp
 245 250 255
 Trp Leu Asp Thr Gln Ser Glu Glu Leu Asn Gln Gln Val Val Ser Ser
 260 265 270
 Ser Glu Gln Leu Gln Ser Cys Gln Ala Glu Ile Ile Glu Leu Arg Arg
 275 280 285
 Thr Val Asn Ala Leu Glu Ile Glu Leu Gln Ala Gln His Ser Met Arg
 290 295 300
 Asp Ala Leu Glu Ser Thr Leu Ala Glu Thr Glu Ala Arg Tyr Ser Ser
 305 310 315 320
 Gln Leu Ala Gln Met Gln Cys Met Ile Thr Asn Val Glu Ala Gln Leu
 325 330 335
 Ala Glu Ile Arg Ala Asp Leu Glu Arg Gln Asn Gln Glu Tyr Gln Val
 340 345 350
 Leu Leu Asp Val Arg Ala Arg Leu Glu Cys Glu Ile Asn Thr Tyr Arg
 355 360 365
 Gly Leu Leu Glu Ser Glu Asp Ser Lys Leu Pro Cys Asn Pro Cys Ala
 370 375 380

Pro Asp Tyr Ser Pro Ser Lys Ser Cys Leu Pro Cys Leu Pro Ala Ala
 385 390 395 400
 Ser Cys Gly Pro Ser Ala Ala Arg Thr Asn Cys Ser Pro Arg Pro Ile
 405 410 415
 Cys Val Pro Cys Pro Gly Gly Arg Phe
 420 425

<210> 434

<211> 467

<212> PRT

<213> Homo sapiens

<400> 434

Met Ala Thr Gln Thr Cys Thr Pro Thr Phe Ser Thr Gly Ser Ile Lys
 1 5 10 15
 Gly Leu Cys Gly Thr Ala Gly Gly Ile Ser Arg Val Ser Ser Ile Arg
 20 25 30
 Ser Val Gly Ser Cys Arg Val Pro Ser Leu Ala Gly Ala Gly Tyr
 35 40 45
 Ile Ser Ser Ala Arg Ser Gly Leu Ser Gly Leu Gly Ser Cys Leu Pro
 50 55 60
 Gly Ser Tyr Leu Ser Ser Glu Cys His Thr Ser Gly Phe Val Gly Ser
 65 70 75 80
 Gly Gly Trp Phe Cys Glu Gly Ser Phe Asn Gly Ser Glu Lys Glu Thr
 85 90 95
 Met Gln Phe Leu Asn Asp Arg Leu Ala Asn Tyr Leu Glu Lys Val Arg
 100 105 110
 Gln Leu Glu Arg Glu Asn Ala Glu Leu Glu Ser Arg Ile Gln Glu Trp
 115 120 125
 Tyr Glu Phe Gln Ile Pro Tyr Ile Cys Pro Asp Tyr Gln Ser Tyr Phe
 130 135 140
 Lys Thr Ile Glu Asp Phe Gln Gln Lys Ile Leu Leu Thr Lys Ser Glu
 145 150 155 160
 Asn Ala Arg Leu Val Leu Gln Ile Asp Asn Ala Lys Leu Ala Ala Asp
 165 170 175
 Asp Phe Arg Thr Lys Tyr Glu Thr Glu Leu Ser Leu Arg Gln Leu Val
 180 185 190
 Glu Ala Asp Ile Asn Gly Leu Arg Arg Ile Leu Asp Glu Leu Thr Leu
 195 200 205
 Cys Lys Ala Asp Leu Glu Ala Gln Val Glu Ser Leu Lys Glu Glu Leu
 210 215 220
 Met Cys Leu Lys Lys Asn His Glu Glu Glu Val Ser Val Leu Arg Cys
 225 230 235 240
 Gln Leu Gly Asp Arg Leu Asn Val Glu Val Asp Ala Ala Pro Pro Val
 245 250 255
 Asp Leu Asn Lys Ile Leu Glu Asp Met Arg Cys Gln Tyr Glu Ala Leu
 260 265 270
 Val Glu Asn Asn Arg Arg Asp Val Glu Ala Trp Phe Asn Thr Gln Thr
 275 280 285
 Glu Glu Leu Asn Gln Gln Val Val Ser Ser Ser Glu Gln Leu Gln Cys
 290 295 300
 Cys Gln Thr Glu Ile Ile Glu Leu Arg Arg Thr Val Asn Ala Leu Glu
 305 310 315 320
 Ile Glu Leu Gln Ala Gln His Ser Met Arg Asn Ser Leu Glu Ser Thr
 325 330 335
 Leu Ala Glu Thr Glu Ala Arg Tyr Ser Ser Gln Leu Ala Gln Met Gln
 340 345 350
 Cys Leu Ile Ser Asn Val Glu Ala Gln Leu Ser Glu Ile Arg Cys Asp
 355 360 365
 Leu Glu Arg Gln Asn Gln Glu Tyr Gln Val Leu Leu Asp Val Lys Ala
 370 375 380

Arg Leu Glu Gly Glu Ile Ala Thr Tyr Arg His Leu Leu Glu Gly Glu
 385 390 395 400
 Asp Cys Lys Leu Pro Gln Pro Cys Ala Thr Ala Cys Lys Pro Val
 405 410 415
 Ile Arg Val Pro Ser Val Pro Pro Val Pro Cys Val Pro Ser Val Pro
 420 425 430
 Cys Thr Pro Ala Pro Gln Val Gly Thr Gln Ile Arg Thr Ile Thr Glu
 435 440 445
 Glu Ile Arg Asp Gly Lys Val Ile Ser Ser Arg Glu His Val Gln Ser
 450 455 460
 Arg Pro Leu
 465

<210> 435

<211> 420

<212> PRT

<213> Homo sapiens

<400> 435

Met Ser Leu Arg Leu Gln Ser Ser Ser Ala Ser Tyr Gly Gly Gly Phe
 1 5 10 15
 Gly Gly Gly Ser Cys Gln Leu Gly Gly Gly Arg Gly Val Ser Thr Cys
 20 25 30
 Ser Thr Arg Phe Val Ser Gly Gly Ser Ala Gly Gly Tyr Gly Gly Gly
 35 40 45
 Val Ser Cys Gly Phe Gly Gly Gly Ala Gly Ser Gly Phe Gly Gly Gly
 50 55 60
 Tyr Gly Gly Gly Leu Gly Gly Tyr Gly Gly Gly Leu Gly Gly Gly
 65 70 75 80
 Phe Gly Gly Gly Phe Ala Gly Gly Phe Val Asp Phe Gly Ala Cys Asp
 85 90 95
 Gly Gly Leu Leu Thr Gly Asn Glu Lys Ile Thr Met Gln Asn Leu Asn
 100 105 110
 Asp Arg Leu Ala Ser Tyr Leu Glu Lys Val Arg Ala Leu Glu Glu Ala
 115 120 125
 Asn Ala Asp Leu Glu Val Lys Ile Arg Asp Trp His Leu Lys Gln Ser
 130 135 140
 Pro Ala Ser Pro Glu Arg Asp Tyr Ser Pro Tyr Tyr Lys Thr Ile Glu
 145 150 155 160
 Glu Leu Arg Asp Lys Ile Leu Thr Ala Thr Ile Glu Asn Asn Arg Val
 165 170 175
 Ile Leu Glu Ile Asp Asn Ala Arg Leu Ala Val Asp Asp Phe Arg Leu
 180 185 190
 Lys Tyr Glu Asn Glu Leu Ala Leu Arg Gln Ser Val Glu Ala Asp Ile
 195 200 205
 Asn Gly Leu Arg Arg Val Leu Asp Glu Leu Thr Leu Ser Lys Thr Asp
 210 215 220
 Leu Glu Met Gln Ile Glu Ser Leu Asn Glu Glu Leu Ala Tyr Met Lys
 225 230 235 240
 Lys Asn His Glu Glu Met Lys Glu Phe Ser Asn Gln Val Val Gly
 245 250 255
 Gln Val Asn Val Glu Met Asp Ala Thr Pro Gly Ile Asp Leu Thr Arg
 260 265 270
 Val Leu Ala Glu Met Arg Glu Gln Tyr Glu Ala Met Ala Glu Arg Asn
 275 280 285
 Arg Arg Asp Ala Glu Glu Trp Phe His Ala Lys Ser Ala Glu Leu Asn
 290 295 300
 Lys Glu Val Ser Thr Asn Thr Ala Met Ile Gln Thr Ser Lys Thr Glu
 305 310 315 320
 Ile Thr Glu Leu Arg Arg Thr Leu Gln Gly Leu Glu Ile Glu Leu Gln
 325 330 335

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Ser Gln Leu Ser Met Lys Ala Gly Leu Glu Asn Thr Val Ala Glu Thr
 340 345 350
 Glu Cys Arg Tyr Ala Leu Gln Leu Gln Ile Gln Gly Leu Ile Ser
 355 360 365
 Ser Ile Glu Ala Gln Leu Ser Glu Leu Arg Ser Glu Met Glu Cys Gln
 370 375 380
 Asn Gln Glu Tyr Lys Met Leu Leu Asp Ile Lys Thr Arg Leu Glu Gln
 385 390 395 400
 Glu Ile Ala Thr Tyr Arg Ser Leu Leu Glu Gly Gln Asp Ala Lys Lys
 405 410 415
 Arg Gln Pro Pro
 420

<210> 436

<211> 456

<212> PRT

<213> Homo sapiens

<400> 436

Met Thr Thr Thr Phe Leu Gln Thr Ser Ser Ser Thr Phe Gly Gly Gly
 1 5 10 15
 Ser Thr Arg Gly Ser Leu Leu Ala Gly Gly Gly Gly Phe Gly Gly
 20 25 30
 Gly Ser Leu Ser Gly Gly Gly Gly Ser Arg Ser Ile Ser Ala Ser Ser
 35 40 45
 Ala Arg Phe Val Ser Ser Gly Ser Gly Gly Gly Tyr Gly Gly Gly Met
 50 55 60
 Arg Val Cys Gly Phe Gly Gly Gly Ala Gly Ser Val Phe Gly Gly Gly
 65 70 75 80
 Phe Gly Gly Gly Val Gly Gly Gly Phe Gly Gly Gly Phe Gly Gly Gly
 85 90 95
 Asp Gly Gly Leu Leu Ser Gly Asn Glu Lys Ile Thr Met Gln Asn Leu
 100 105 110
 Asn Asp Arg Leu Ala Ser Tyr Leu Asp Lys Val Arg Ala Leu Glu Glu
 115 120 125
 Ala Asn Ala Asp Leu Glu Val Lys Ile His Asp Trp Tyr Gln Lys Gln
 130 135 140
 Thr Pro Thr Ser Pro Glu Cys Asp Tyr Ser Gln Tyr Phe Lys Thr Ile
 145 150 155 160
 Glu Glu Leu Arg Asp Lys Ile Met Ala Thr Thr Ile Asp Asn Ser Arg
 165 170 175
 Val Ile Leu Glu Ile Asp Asn Ala Arg Leu Ala Ala Asp Asp Phe Arg
 180 185 190
 Leu Lys Tyr Glu Asn Glu Leu Ala Leu Arg Gln Gly Val Glu Ala Asp
 195 200 205
 Ile Asn Gly Leu Arg Arg Val Leu Asp Glu Leu Thr Leu Ala Arg Thr
 210 215 220
 Asp Leu Glu Met Gln Ile Glu Gly Leu Asn Glu Glu Leu Ala Tyr Leu
 225 230 235 240
 Lys Lys Asn His Glu Glu Glu Met Lys Glu Phe Ser Ser Gln Leu Ala
 245 250 255
 Gly Gln Val Asn Val Glu Met Asp Ala Ala Pro Gly Val Asp Leu Thr
 260 265 270
 Arg Val Leu Ala Glu Met Arg Glu Gln Tyr Glu Ala Met Ala Glu Lys
 275 280 285
 Asn Arg Arg Asp Val Glu Ala Trp Phe Phe Ser Lys Thr Glu Glu Leu
 290 295 300
 Asn Lys Glu Val Ala Ser Asn Thr Glu Met Ile Gln Thr Ser Lys Thr
 305 310 315 320
 Glu Ile Thr Asp Leu Arg Arg Thr Met Gln Glu Leu Glu Ile Glu Leu
 325 330 335

Gln Ser Gln Leu Ser Met Lys Ala Gly Leu Glu Asn Ser Leu Ala Glu
 340 345 350
 Thr Glu Cys Arg Tyr Ala Thr Gln Leu Gln Gln Ile Gln Gly Leu Ile
 355 360 365
 Gly Gly Leu Glu Ala Gln Leu Ser Glu Leu Arg Cys Glu Met Glu Ala
 370 375 380
 Gln Asn Gln Glu Tyr Lys Met Leu Leu Asp Ile Lys Thr Arg Leu Glu
 385 390 395 400
 Gln Glu Ile Ala Thr Tyr Arg Ser Leu Leu Glu Gly Gln Asp Ala Lys
 405 410 415
 Met Ala Gly Ile Gly Ile Arg Glu Ala Ser Ser Gly Gly Gly Ser
 420 425 430
 Ser Ser Asn Phe His Ile Asn Val Glu Glu Ser Val Asp Gly Gln Val
 435 440 445
 Val Ser Ser His Lys Arg Glu Ile
 450 455
 <210> 437
 <211> 400
 <212> PRT
 <213> Homo sapiens

<400> 437
 Met Thr Ser Tyr Ser Tyr Arg Gln Ser Ser Ala Thr Ser Ser Phe Gly
 1 5 10 15
 Gly Leu Gly Gly Gly Ser Val Arg Phe Gly Pro Gly Val Ala Phe Arg
 20 25 30
 Ala Pro Ser Ile His Gly Gly Ser Gly Arg Gly Val Ser Val Ser
 35 40 45
 Ser Ala Arg Phe Val Ser Ser Ser Ser Gly Ala Tyr Gly Gly Gly
 50 55 60
 Tyr Gly Gly Val Leu Thr Ala Ser Asp Gly Leu Leu Ala Gly Asn Glu
 65 70 75 80
 Lys Leu Thr Met Gln Asn Leu Asn Asp Arg Leu Ala Ser Tyr Leu Asp
 85 90 95
 Lys Val Arg Ala Leu Glu Ala Ala Asn Gly Glu Leu Glu Val Lys Ile
 100 105 110
 Arg Asp Trp Tyr Gln Lys Gln Gly Pro Gly Pro Ser Arg Asp Tyr Ser
 115 120 125
 His Tyr Tyr Thr Thr Ile Gln Asp Leu Arg Asp Lys Ile Leu Gly Ala
 130 135 140
 Thr Ile Glu Asn Ser Arg Ile Val Leu Gln Ile Asp Asn Ala Arg Leu
 145 150 155 160
 Ala Ala Asp Asp Phe Arg Thr Lys Phe Glu Thr Glu Gln Ala Leu Arg
 165 170 175
 Met Ser Val Glu Ala Asp Ile Asn Gly Leu Arg Arg Val Leu Asp Glu
 180 185 190
 Leu Thr Leu Ala Arg Thr Asp Leu Glu Met Gln Ile Glu Gly Leu Lys
 195 200 205
 Glu Glu Leu Ala Tyr Leu Lys Lys Asn His Glu Glu Glu Ile Ser Thr
 210 215 220
 Leu Arg Gly Gln Val Gly Gly Gln Val Ser Val Glu Val Asp Ser Ala
 225 230 235 240
 Pro Gly Thr Asp Leu Ala Lys Ile Leu Ser Asp Met Arg Ser Gln Tyr
 245 250 255
 Glu Val Met Ala Glu Gln Asn Arg Lys Asp Ala Glu Ala Trp Phe Thr
 260 265 270
 Ser Arg Thr Glu Glu Leu Asn Arg Glu Val Ala Gly His Thr Glu Gln
 275 280 285
 Leu Gln Met Ser Arg Ser Glu Val Thr Asp Leu Arg Arg Thr Leu Gln
 290 295 300

Gly Leu Glu Ile Glu Leu Gln Ser Gln Leu Ser Met Lys Ala Ala Leu
 305 310 315 320
 Glu Asp Thr Leu Ala Glu Thr Glu Ala Arg Phe Gly Ala Gln Leu Ala
 325 330 335
 His Ile Gln Ala Leu Ile Ser Gly Ile Glu Ala Gln Leu Gly Asp Val
 340 345 350
 Arg Ala Asp Ser Glu Arg Gln Asn Gln Glu Tyr Gln Arg Leu Met Asp
 355 360 365
 Ile Lys Ser Arg Leu Glu Gln Glu Ile Ala Thr Tyr Arg Ser Leu Leu
 370 375 380
 Glu Gly Gln Glu Asp His Tyr Asn Asn Leu Ser Ala Ser Lys Val Leu
 385 390 395 400

<210> 438

<211> 622

<212> PRT

<213> Homo sapiens

<400> 438

Met Ser Cys Arg Gln Phe Ser Ser Ser Tyr Leu Thr Ser Gly Gly Gly
 1 5 10 15
 Gly Gly Gly Gly Leu Gly Ser Gly Gly Ser Ile Arg Ser Ser Tyr Ser
 20 25 30
 Arg Phe Ser Ser Ser Gly Gly Arg Gly Gly Gly Gly Arg Phe Ser Ser
 35 40 45
 Ser Ser Gly Tyr Gly Gly Gly Ser Ser Arg Val Cys Gly Arg Gly Gly
 50 55 60
 Gly Gly Ser Phe Gly Tyr Ser Tyr Gly Gly Gly Ser Gly Gly Gly Phe
 65 70 75 80
 Ser Ala Ser Ser Leu Gly Gly Gly Phe Gly Gly Gly Ser Arg Gly Phe
 85 90 95
 Gly Gly Ala Ser Gly Gly Gly Tyr Ser Ser Ser Gly Gly Phe Gly Gly
 100 105 110
 Gly Phe Gly Gly Gly Ser Gly Gly Phe Gly Gly Gly Tyr Gly Ser
 115 120 125
 Gly Phe Gly Gly Leu Gly Gly Phe Gly Gly Gly Ala Gly Gly Gly Asp
 130 135 140
 Gly Gly Ile Leu Thr Ala Asn Glu Lys Ser Thr Met Gln Glu Leu Asn
 145 150 155 160
 Ser Arg Leu Ala Ser Tyr Leu Asp Lys Val Gln Ala Leu Glu Glu Ala
 165 170 175
 Asn Asn Asp Leu Glu Asn Lys Ile Gln Asp Trp Tyr Asp Lys Lys Gly
 180 185 190
 Pro Ala Ala Ile Gln Lys Asn Tyr Ser Pro Tyr Tyr Asn Thr Ile Asp
 195 200 205
 Asp Leu Lys Asp Gln Ile Val Asp Leu Thr Val Gly Asn Asn Lys Thr
 210 215 220
 Leu Leu Asp Ile Asp Asn Thr Arg Met Thr Leu Asp Asp Phe Arg Ile
 225 230 235 240
 Lys Phe Glu Met Glu Gln Asn Leu Arg Gln Gly Val Asp Ala Asp Ile
 245 250 255
 Asn Gly Leu Arg Gln Val Leu Asp Asn Leu Thr Met Glu Lys Ser Asp
 260 265 270
 Leu Glu Met Gln Tyr Glu Thr Leu Gln Glu Glu Leu Met Ala Leu Lys
 275 280 285
 Lys Asn His Lys Glu Glu Met Ser Gln Leu Thr Gly Gln Asn Ser Gly
 290 295 300
 Asp Val Asn Val Glu Ile Asn Val Ala Pro Gly Lys Asp Leu Thr Lys
 305 310 315 320
 Thr Leu Asn Asp Met Arg Gln Glu Tyr Glu Gln Leu Ile Ala Lys Asn
 325 330 335

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Arg Lys Asp Ile Glu Asn Gln Tyr Glu Thr Gln Ile Thr Gln Ile Glu
 340 345 350
 His Glu Val Ser Ser Ser Gly Gln Glu Val Gln Ser Ser Ala Lys Glu
 355 360 365
 Val Thr Gln Leu Arg His Gly Val Gln Glu Leu Glu Ile Glu Leu Gln
 370 375 380
 Ser Gln Leu Ser Lys Lys Ala Ala Leu Glu Lys Ser Leu Glu Asp Thr
 385 390 395 400
 Lys Asn Arg Tyr Cys Gly Gln Leu Gln Met Ile Gln Glu Gln Ile Ser
 405 410 415
 Asn Leu Glu Ala Gln Ile Thr Asp Val Arg Gln Glu Ile Glu Cys Gln
 420 425 430
 Asn Gln Glu Tyr Ser Leu Leu Leu Ser Ile Lys Met Arg Leu Glu Lys
 435 440 445
 Glu Ile Glu Thr Tyr His Asn Leu Leu Glu Gly Gly Gln Glu Asp Phe
 450 455 460
 Glu Ser Ser Gly Ala Gly Lys Ile Gly Leu Gly Gly Arg Gly Gly Ser
 465 470 475 480
 Gly Gly Ser Tyr Gly Arg Gly Ser Arg Gly Gly Ser Gly Gly Ser Tyr
 485 490 495
 Gly Gly Gly Gly Ser Gly Gly Gly Tyr Gly Gly Gly Ser Gly Ser Arg
 500 505 510
 Gly Gly Ser Gly Gly Ser Tyr Gly Gly Gly Ser Gly Ser Gly Gly Gly
 515 520 525
 Ser Gly Gly Gly Tyr Gly Gly Ser Gly Gly Gly His Ser Gly Gly
 530 535 540
 Ser Gly Gly Gly His Ser Gly Gly Ser Gly Gly Asn Tyr Gly Gly Gly
 545 550 555 560
 Ser Gly Ser Gly Gly Gly Ser Gly Gly Gly Tyr Gly Gly Ser Gly
 565 570 575
 Ser Arg Gly Gly Ser Gly Gly Ser His Gly Gly Gly Ser Gly Phe Gly
 580 585 590
 Gly Glu Ser Gly Gly Ser Tyr Gly Gly Gly Glu Glu Ala Ser Gly Ser
 595 600 605
 Gly Gly Gly Tyr Gly Gly Gly Ser Gly Lys Ser Ser His Ser
 610 615 620
 <210> 439
 <211> 472
 <212> PRT
 <213> Homo sapiens

<400> 439
 Met Thr Thr Cys Ser Arg Gln Phe Thr Ser Ser Ser Ser Met Lys Gly
 1 5 10 15
 Ser Cys Gly Ile Gly Gly Gly Ile Gly Gly Ser Ser Arg Ile Ser
 20 25 30
 Ser Val Leu Ala Gly Gly Ser Cys Arg Ala Pro Ser Thr Tyr Gly Gly
 35 40 45
 Gly Leu Ser Val Ser Ser Ser Arg Phe Ser Ser Gly Gly Ala Tyr Gly
 50 55 60
 Leu Gly Gly Gly Tyr Gly Gly Gly Phe Ser Ser Ser Ser Ser Phe
 65 70 75 80
 Gly Ser Gly Phe Gly Gly Gly Tyr Gly Gly Gly Leu Gly Ala Gly Leu
 85 90 95
 Gly Gly Gly Phe Gly Gly Gly Phe Ala Gly Gly Asp Gly Leu Leu Val
 100 105 110
 Gly Ser Glu Lys Val Thr Met Gln Asn Leu Asn Asp Arg Leu Ala Ser
 115 120 125
 Tyr Leu Asp Lys Val Arg Ala Leu Glu Glu Ala Asn Ala Asp Leu Glu
 130 135 140

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Val Lys Ile Arg Asp Trp Tyr Gln Arg Gln Arg Pro Ala Glu Ile Lys
 145 150 155 160
 Asp Tyr Ser Pro Tyr Phe Lys Thr Ile Glu Asp Leu Arg Asn Lys Ile
 165 170 175
 Leu Thr Ala Thr Val Asp Asn Ala Asn Val Leu Leu Gln Ile Asp Asn
 180 185 190
 Ala Arg Leu Ala Ala Asp Asp Phe Arg Thr Lys Tyr Glu Thr Glu Leu
 195 200 205
 Asn Leu Arg Met Ser Val Glu Ala Asp Ile Asn Gly Leu Arg Arg Val
 210 215 220
 Leu Asp Glu Leu Thr Leu Ala Arg Ala Asp Leu Glu Met Gln Ile Glu
 225 230 235 240
 Ser Leu Lys Glu Glu Leu Ala Tyr Leu Lys Lys Asn His Glu Glu Glu
 245 250 255
 Met Asn Ala Leu Arg Gly Gln Val Gly Gly Asp Val Asn Val Glu Met
 260 265 270
 Asp Ala Ala Pro Gly Val Asp Leu Ser Arg Ile Leu Asn Glu Met Arg
 275 280 285
 Asp Gln Tyr Glu Lys Met Ala Glu Lys Asn Arg Lys Asp Ala Glu Glu
 290 295 300
 Trp Phe Phe Thr Lys Thr Glu Glu Leu Asn Arg Glu Val Ala Thr Asn
 305 310 315 320
 Ser Glu Leu Val Gln Ser Gly Lys Ser Glu Ile Ser Glu Leu Arg Arg
 325 330 335
 Thr Met Gln Asn Leu Glu Ile Glu Leu Gln Ser Gln Leu Ser Met Lys
 340 345 350
 Ala Ser Leu Glu Asn Ser Leu Glu Glu Thr Lys Gly Arg Tyr Cys Met
 355 360 365
 Gln Leu Ala Gln Ile Gln Glu Met Ile Gly Ser Val Glu Glu Gln Leu
 370 375 380
 Ala Gln Leu Arg Cys Glu Met Glu Gln Gln Asn Gln Glu Tyr Lys Ile
 385 390 395 400
 Leu Leu Asp Val Lys Thr Arg Leu Glu Gln Glu Ile Ala Thr Tyr Arg
 405 410 415
 Arg Leu Leu Glu Gly Glu Asp Ala His Leu Ser Ser Ser Gln Phe Ser
 420 425 430
 Ser Gly Ser Gln Ser Ser Arg Asp Val Thr Ser Ser Ser Arg Gln Ile
 435 440 445
 Arg Thr Lys Val Met Asp Val His Asp Gly Lys Val Val Ser Thr His
 450 455 460
 Glu Gln Val Leu Arg Thr Lys Asn
 465 470

<210> 440

<211> 473

<212> PRT

<213> Homo sapiens

<400> 440

Met Thr Thr Cys Ser Arg Gln Phe Thr Ser Ser Ser Ser Met Lys Gly
 1 5 10 15
 Ser Cys Gly Ile Gly Gly Gly Ile Gly Gly Gly Ser Ser Arg Ile Ser
 20 25 30
 Ser Val Leu Ala Gly Gly Ser Cys Arg Ala Pro Ser Thr Tyr Gly Gly
 35 40 45
 Gly Leu Ser Val Ser Ser Arg Phe Ser Ser Gly Gly Ala Cys Gly Leu
 50 55 60
 Gly Gly Gly Tyr Gly Gly Gly Phe Ser Ser Ser Ser Phe Gly Ser
 65 70 75 80
 Gly Phe Gly Gly Gly Tyr Gly Gly Gly Leu Gly Ala Gly Phe Gly Gly
 85 90 95

Gly Leu Gly Ala Gly Phe Gly Gly Gly Phe Ala Gly Gly Asp Gly Leu
 100 105 110
 Leu Val Gly Ser Glu Lys Val Thr Met Gln Asn Leu Asn Asp Arg Leu
 115 120 125
 Ala Ser Tyr Leu Asp Lys Val Arg Ala Leu Glu Glu Ala Asn Ala Asp
 130 135 140
 Leu Glu Val Lys Ile Arg Asp Trp Tyr Gln Arg Gln Arg Pro Ser Glu
 145 150 155 160
 Ile Lys Asp Tyr Ser Pro Tyr Phe Lys Thr Ile Glu Asp Leu Arg Asn
 165 170 175
 Lys Ile Ile Ala Ala Thr Ile Glu Asn Ala Gln Pro Ile Leu Gln Ile
 180 185 190
 Asp Asn Ala Arg Leu Ala Ala Asp Asp Phe Arg Thr Lys Tyr Glu His
 195 200 205
 Glu Leu Ala Leu Arg Gln Thr Val Glu Ala Asp Val Asn Gly Leu Arg
 210 215 220
 Arg Val Leu Asp Glu Leu Thr Leu Ala Arg Thr Asp Leu Glu Met Gln
 225 230 235 240
 Ile Glu Gly Leu Lys Glu Glu Leu Ala Tyr Leu Arg Lys Asn His Glu
 245 250 255
 Glu Glu Met Leu Ala Leu Arg Gly Gln Thr Gly Gly Asp Val Asn Val
 260 265 270
 Glu Met Asp Ala Ala Pro Gly Val Asp Leu Ser Arg Ile Leu Asn Glu
 275 280 285
 Met Arg Asp Gln Tyr Glu Gln Met Ala Glu Lys Asn Arg Arg Asp Ala
 290 295 300
 Glu Thr Trp Phe Leu Ser Lys Thr Glu Glu Leu Asn Lys Glu Val Ala
 305 310 315 320
 Ser Asn Ser Glu Leu Val Gln Ser Ser Arg Ser Glu Val Thr Glu Leu
 325 330 335
 Arg Arg Val Leu Gln Gly Leu Glu Ile Glu Leu Gln Ser Gln Leu Ser
 340 345 350
 Met Lys Ala Ser Leu Glu Asn Ser Leu Glu Glu Thr Lys Gly Arg Tyr
 355 360 365
 Cys Met Gln Leu Ser Gln Ile Gln Gly Leu Ile Gly Ser Val Glu Glu
 370 375 380
 Gln Leu Ala Gln Leu Arg Cys Glu Met Glu Gln Gln Ser Gln Glu Tyr
 385 390 395 400
 Gln Ile Leu Leu Asp Val Lys Thr Arg Leu Glu Gln Glu Ile Ala Thr
 405 410 415
 Tyr Arg Arg Leu Leu Glu Gly Glu Asp Ala His Leu Ser Ser Gln Gln
 420 425 430
 Ala Ser Gly Gln Ser Tyr Ser Ser Arg Glu Val Phe Thr Ser Ser Ser
 435 440 445
 Ser Ser Ser Ser Arg Gln Thr Arg Pro Ile Leu Lys Glu Gln Ser Ser
 450 455 460
 Ser Ser Phe Ser Gln Gly Gln Ser Ser
 465 470
 <210> 441
 <211> 432
 <212> PRT
 <213> Homo sapiens
 <400> 441
 Met Thr Thr Ser Ile Arg Gln Phe Thr Ser Ser Ser Ser Ile Lys Gly
 1 5 10 15
 Ser Ser Gly Leu Gly Gly Gly Ser Ser Arg Thr Ser Cys Arg Leu Ser
 20 25 30
 Gly Gly Leu Gly Ala Gly Ser Cys Arg Leu Gly Ser Ala Gly Gly Leu
 35 40 45

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Gly Ser Thr Leu Gly Gly Ser Ser Tyr Ser Ser Cys Tyr Ser Phe Gly
 50 55 60
 Ser Gly Gly Gly Tyr Gly Ser Ser Phe Gly Gly Val Asp Gly Leu Leu
 65 70 75 80
 Ala Gly Gly Glu Lys Ala Thr Met Gln Asn Leu Asn Asp Arg Leu Ala
 85 90 95
 Ser Tyr Leu Asp Lys Val Arg Ala Leu Glu Glu Ala Asn Thr Glu Leu
 100 105 110
 Glu Val Lys Ile Arg Asp Trp Tyr Gln Arg Gln Ala Pro Gly Pro Ala
 115 120 125
 Arg Asp Tyr Ser Gln Tyr Tyr Arg Thr Ile Glu Glu Leu Gln Asn Lys
 130 135 140
 Ile Leu Thr Ala Thr Val Asp Asn Ala Asn Ile Leu Leu Gln Ile Asp
 145 150 155 160
 Asn Ala Arg Leu Ala Ala Asp Asp Phe Arg Thr Lys Phe Glu Thr Glu
 165 170 175
 Gln Ala Leu Arg Leu Ser Val Glu Ala Asp Ile Asn Gly Leu Arg Arg
 180 185 190
 Val Leu Asp Glu Leu Thr Leu Ala Arg Ala Asp Leu Glu Met Gln Ile
 195 200 205
 Glu Asn Leu Lys Glu Glu Leu Ala Tyr Leu Lys Lys Asn His Glu Glu
 210 215 220
 Glu Met Asn Ala Leu Arg Gly Gln Val Gly Gly Glu Ile Asn Val Glu
 225 230 235 240
 Met Asp Ala Ala Pro Gly Val Asp Leu Ser Arg Ile Leu Asn Glu Met
 245 250 255
 Arg Asp Gln Tyr Glu Lys Met Ala Glu Lys Asn Arg Lys Asp Ala Glu
 260 265 270
 Asp Trp Phe Phe Ser Lys Thr Glu Glu Leu Asn Arg Glu Val Ala Thr
 275 280 285
 Asn Ser Glu Leu Val Gln Ser Gly Lys Ser Glu Ile Ser Glu Leu Arg
 290 295 300
 Arg Thr Met Gln Ala Leu Glu Ile Glu Leu Gln Ser Gln Leu Ser Met
 305 310 315 320
 Lys Ala Ser Leu Glu Gly Asn Leu Ala Glu Thr Glu Asn Arg Tyr Cys
 325 330 335
 Val Gln Leu Ser Gln Ile Gln Gly Leu Ile Gly Ser Val Glu Glu Gln
 340 345 350
 Leu Ala Gln Leu Arg Cys Glu Met Glu Gln Gln Asn Gln Glu Tyr Lys
 355 360 365
 Ile Leu Leu Asp Val Lys Thr Arg Leu Glu Gln Glu Ile Ala Thr Tyr
 370 375 380
 Arg Arg Leu Leu Glu Gly Asp Ala His Leu Thr Gln Tyr Lys Lys
 385 390 395 400
 Glu Pro Val Thr Thr Arg Gln Val Arg Thr Ile Val Glu Glu Val Gln
 405 410 415
 Asp Gly Lys Val Ile Ser Ser Arg Glu Gln Val His Gln Thr Thr Arg
 420 425 430
 <210> 442
 <211> 469
 <212> PRT
 <213> Homo sapiens

 <400> 442
 Met Ser Ile His Phe Ser Ser Pro Val Phe Thr Ser Arg Ser Ala Ala
 1 5 10 15
 Phe Ser Gly Arg Gly Ala Gln Val Arg Leu Ser Ser Ala Arg Pro Gly
 20 25 30
 Gly Leu Gly Ser Ser Ser Leu Tyr Gly Leu Gly Ala Ser Arg Pro Arg
 35 40 45

Val Ala Val Arg Ser Ala Tyr Gly Gly Pro Val Gly Ala Gly Ile Arg
 50 55 60
 Glu Val Thr Ile Asn Gln Ser Leu Leu Ala Pro Leu Arg Leu Asp Ala
 65 70 75 80
 Asp Pro Ser Leu Gln Arg Val Arg Gln Glu Glu Ser Glu Gln Ile Lys
 85 90 95
 Thr Leu Asn Asn Lys Phe Ala Ser Phe Ile Asp Lys Val Arg Phe Leu
 100 105 110
 Glu Gln Gln Asn Lys Leu Leu Glu Thr Lys Trp Thr Leu Leu Gln Glu
 115 120 125
 Gln Lys Ser Ala Lys Ser Ser Arg Leu Pro Asp Ile Phe Glu Ala Gln
 130 135 140
 Ile Ala Gly Leu Arg Gly Gln Leu Glu Ala Leu Gln Val Asp Gly Gly
 145 150 155 160
 Arg Leu Glu Ala Glu Leu Arg Ser Met Gln Asp Val Val Glu Asp Phe
 165 170 175
 Lys Asn Lys Tyr Glu Asp Glu Ile Asn Arg Arg Thr Ala Ala Glu Asn
 180 185 190
 Glu Phe Val Val Leu Lys Lys Asp Val Asp Ala Ala Tyr Met Ser Lys
 195 200 205
 Val Glu Leu Glu Ala Lys Val Asp Ala Leu Asn Asp Glu Ile Asn Phe
 210 215 220
 Leu Arg Thr Leu Asn Glu Thr Glu Leu Thr Glu Leu Gln Ser Gln Ile
 225 230 235 240
 Ser Asp Thr Ser Val Leu Ser Met Asp Asn Ser Arg Ser Leu Asp
 245 250 255
 Leu Asp Gly Ile Ile Ala Glu Val Lys Ala Gln Tyr Glu Glu Met Ala
 260 265 270
 Lys Cys Ser Arg Ala Glu Ala Glu Ala Trp Tyr Gln Thr Lys Phe Glu
 275 280 285
 Thr Leu Gln Ala Gln Ala Gly Lys His Gly Asp Asp Leu Arg Asn Thr
 290 295 300
 Arg Asn Glu Ile Ser Glu Met Asn Arg Ala Ile Gln Arg Leu Gln Ala
 305 310 315 320
 Glu Ile Asp Asn Ile Lys Asn Gln Arg Ala Lys Leu Glu Ala Ala Ile
 325 330 335
 Ala Glu Ala Glu Glu Arg Gly Glu Leu Ala Leu Lys Asp Ala Arg Ala
 340 345 350
 Lys Gln Glu Glu Leu Glu Ala Ala Leu Gln Arg Ala Lys Gln Asp Met
 355 360 365
 Ala Arg Gln Leu Arg Glu Tyr Gln Glu Leu Met Ser Val Lys Leu Ala
 370 375 380
 Leu Asp Ile Glu Ile Ala Thr Tyr Arg Lys Leu Leu Glu Gly Glu Glu
 385 390 395 400
 Ser Arg Leu Ala Gly Asp Gly Val Gly Ala Val Asn Ile Ser Val Met
 405 410 415
 Asn Ser Thr Gly Gly Ser Ser Ser Gly Gly Gly Ile Gly Leu Thr Leu
 420 425 430
 Gly Gly Thr Met Gly Ser Asn Ala Leu Ser Phe Ser Ser Ser Ala Gly
 435 440 445
 Pro Gly Leu Leu Lys Ala Tyr Ser Ile Arg Thr Ala Ser Ala Ser Arg
 450 455 460
 Arg Ser Ala Arg Asp
 465

<210> 443

<211> 486

<212> PRT

<213> Homo sapiens

<400> 443

Met	Thr	Cys	Gly	Ser	Tyr	Cys	Gly	Gly	Arg	Ala	Phe	Ser	Cys	Ile	Ser
1				5					10					15	
Ala	Cys	Gly	Pro	Arg	Pro	Gly	Arg	Cys	Cys	Ile	Thr	Ala	Ala	Pro	Tyr
			20					25					30		
Arg	Gly	Ile	Ser	Cys	Tyr	Arg	Gly	Leu	Thr	Gly	Gly	Phe	Gly	Ser	His
		35					40					45			
Ser	Val	Cys	Gly	Gly	Phe	Arg	Ala	Gly	Ser	Cys	Gly	Arg	Ser	Phe	Gly
	50					55					60				
Tyr	Arg	Ser	Gly	Gly	Val	Cys	Gly	Pro	Ser	Pro	Pro	Cys	Ile	Thr	Thr
65					70					75					80
Val	Ser	Val	Asn	Glu	Ser	Leu	Leu	Thr	Pro	Leu	Asn	Leu	Glu	Ile	Asp
			85						90					95	
Pro	Asn	Ala	Gln	Cys	Val	Lys	Gln	Glu	Lys	Glu	Gln	Ile	Lys	Ser	
			100					105					110		
Leu	Asn	Ser	Arg	Phe	Ala	Ala	Phe	Ile	Asp	Lys	Val	Arg	Phe	Leu	Glu
		115					120					125			
Gln	Gln	Asn	Lys	Leu	Leu	Glu	Thr	Lys	Leu	Gln	Phe	Tyr	Gln	Asn	Arg
		130				135					140				
Glu	Cys	Cys	Gln	Ser	Asn	Leu	Glu	Pro	Leu	Phe	Glu	Gly	Tyr	Ile	Glu
145					150					155					160
Thr	Leu	Arg	Arg	Glu	Ala	Glu	Cys	Val	Glu	Ala	Asp	Ser	Gly	Arg	Leu
			165					170						175	
Ala	Ser	Glu	Leu	Asn	His	Val	Gln	Glu	Val	Leu	Glu	Gly	Tyr	Lys	Lys
			180					185					190		
Lys	Tyr	Glu	Glu	Glu	Val	Ser	Leu	Arg	Ala	Thr	Ala	Glu	Asn	Glu	Phe
		195					200					205			
Val	Ala	Leu	Lys	Lys	Asp	Val	Asp	Cys	Ala	Tyr	Leu	Arg	Lys	Ser	Asp
	210					215					220				
Leu	Glu	Ala	Asn	Val	Glu	Ala	Leu	Ile	Gln	Glu	Ile	Asp	Phe	Leu	Arg
225					230					235					240
Arg	Leu	Tyr	Glu	Glu	Glu	Ile	Arg	Val	Leu	Gln	Ser	His	Ile	Ser	Asp
			245					250					255		
Thr	Ser	Val	Val	Val	Lys	Leu	Asp	Asn	Ser	Arg	Asp	Leu	Asn	Met	Asp
			260					265					270		
Cys	Ile	Ile	Ala	Glu	Ile	Lys	Ala	Gln	Tyr	Asp	Asp	Ile	Val	Thr	Arg
		275					280					285			
Ser	Arg	Ala	Glu	Ala	Glu	Ser	Trp	Tyr	Arg	Ser	Lys	Cys	Glu	Glu	Met
	290					295					300				
Lys	Ala	Thr	Val	Ile	Arg	His	Gly	Glu	Thr	Leu	Arg	Arg	Thr	Lys	Glu
305					310					315					320
Glu	Ile	Asn	Glu	Leu	Asn	Arg	Met	Ile	Gln	Arg	Leu	Thr	Ala	Glu	Val
			325						330					335	
Glu	Asn	Ala	Lys	Cys	Gln	Asn	Ser	Lys	Leu	Glu	Ala	Ala	Val	Ala	Gln
		340					345						350		
Ser	Glu	Gln	Gln	Gly	Glu	Ala	Ala	Leu	Ser	Asp	Ala	Arg	Cys	Lys	Leu
		355					360					365			
Ala	Glu	Leu	Glu	Gly	Ala	Leu	Gln	Lys	Ala	Lys	Gln	Asp	Met	Ala	Cys
	370					375					380				
Leu	Ile	Arg	Glu	Tyr	Gln	Glu	Val	Met	Asn	Ser	Lys	Leu	Gly	Leu	Asp
385					390					395					400
Ile	Glu	Ile	Ala	Thr	Tyr	Arg	Arg	Leu	Leu	Glu	Gly	Glu	Glu	Gln	Arg
			405					410						415	
Leu	Cys	Glu	Gly	Val	Gly	Ser	Val	Asn	Val	Cys	Val	Ser	Ser	Ser	Arg
			420					425					430		
Gly	Gly	Val	Val	Cys	Gly	Asp	Leu	Cys	Ala	Ser	Thr	Thr	Ala	Pro	Val
		435					440					445			
Val	Ser	Thr	Arg	Val	Ser	Ser	Val	Pro	Ser	Asn	Ser	Asn	Val	Val	Val
	450					455					460				
Gly	Thr	Thr	Asn	Ala	Cys	Ala	Pro	Ser	Ala	Arg	Val	Gly	Val	Cys	Gly
465					470					475					480
Gly	Ser	Cys	Lys	Arg	Cys										
				485											

<210> 444

<211> 111

<212> PRT

<213> Homo sapiens

<400> 444

Met	Lys	Ala	Thr	Val	Ile	Trp	His	Gly	Glu	Thr	Val	Gly	Cys	Thr	Lys
1				5					10					15	
Glu	Glu	Ile	Lys	Glu	Leu	Thr	His	Met	Ile	Gln	Arg	Leu	Met	Ala	Lys
			20					25					30		
Val	Glu	Asn	Ala	Lys	Cys	Gln	Val	Trp	Gly	Ile	Cys	Ala	Gln	Gly	Gln
		35					40					45			
Arg	Asp	Leu	Trp	Pro	Asn	Leu	Cys	His	Thr	Ala	Tyr	Val	Cys	Pro	Thr
	50				55						60				
Trp	Ile	Ser	Ala	Phe	Ile	Leu	Gln	Ser	Leu	Cys	Pro	Cys	Arg	Val	Pro
65					70				75						80
Gly	Cys	Gly	Gln	Ser	Gly	Ser	Ala	Arg	Met	Met	Lys	Ala	Arg	Gly	Leu
			85					90						95	
Phe	Leu	Arg	Cys	Ser	Gln	Leu	Asn	Gly	Arg	Leu	Asp	Ile	Phe	Arg	
			100					105						110	

<210> 445

<211> 505

<212> PRT

<213> Homo sapiens

<400> 445

Met	Thr	Cys	Gly	Ser	Gly	Phe	Gly	Gly	Arg	Ala	Phe	Ser	Cys	Ile	Ser
1				5					10					15	
Ala	Cys	Gly	Pro	Arg	Pro	Gly	Arg	Cys	Cys	Ile	Thr	Ala	Ala	Pro	Tyr
			20					25					30		
Arg	Gly	Ile	Ser	Cys	Tyr	Arg	Gly	Leu	Thr	Gly	Gly	Phe	Gly	Ser	His
		35					40					45			
Ser	Val	Cys	Gly	Gly	Phe	Arg	Ala	Gly	Ser	Cys	Gly	Arg	Ser	Phe	Gly
	50				55					60					
Tyr	Arg	Ser	Gly	Gly	Val	Cys	Gly	Pro	Ser	Pro	Pro	Cys	Ile	Thr	Thr
65					70				75						80
Val	Ser	Val	Asn	Glu	Ser	Leu	Leu	Thr	Pro	Leu	Asn	Leu	Glu	Ile	Asp
			85						90					95	
Pro	Asn	Ala	Gln	Cys	Val	Lys	Gln	Glu	Glu	Lys	Glu	Gln	Ile	Lys	Ser
			100					105					110		
Leu	Asn	Ser	Arg	Phe	Ala	Ala	Phe	Ile	Asp	Lys	Val	Arg	Phe	Leu	Glu
		115					120					125			
Gln	Gln	Asn	Lys	Leu	Leu	Glu	Thr	Lys	Leu	Gln	Phe	Tyr	Gln	Asn	Arg
	130					135					140				
Glu	Cys	Cys	Gln	Ser	Asn	Leu	Glu	Pro	Leu	Phe	Glu	Gly	Tyr	Ile	Glu
145					150					155					160
Thr	Leu	Arg	Arg	Glu	Ala	Glu	Cys	Val	Glu	Ala	Asp	Ser	Gly	Arg	Leu
				165					170					175	
Ala	Ser	Glu	Leu	Asn	His	Val	Gln	Glu	Val	Leu	Glu	Gly	Tyr	Lys	Lys
		180						185					190		
Lys	Tyr	Glu	Glu	Glu	Val	Ser	Leu	Arg	Ala	Thr	Ala	Glu	Asn	Glu	Phe
		195				200						205			
Val	Ala	Leu	Lys	Lys	Asp	Val	Asp	Cys	Ala	Tyr	Leu	Arg	Lys	Ser	Asp
	210					215						220			
Leu	Glu	Ala	Asn	Val	Glu	Ala	Leu	Ile	Gln	Glu	Ile	Asp	Phe	Leu	Arg
225					230					235					240
Arg	Leu	Tyr	Glu	Glu	Glu	Ile	Arg	Ile	Leu	Gln	Ser	His	Ile	Ser	Asp
				245					250						255

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Thr Ser Val Val Val Lys Leu Asp Asn Ser Arg Asp Leu Asn Met Asp
 260 265 270
 Cys Ile Ile Ala Glu Ile Lys Ala Gln Tyr Asp Asp Ile Val Thr Arg
 275 280 285
 Ser Arg Ala Glu Ala Glu Ser Trp Tyr Arg Ser Lys Cys Glu Glu Met
 290 295 300
 Lys Ala Thr Val Ile Arg His Gly Glu Thr Leu Arg Arg Thr Lys Glu
 305 310 315 320
 Glu Ile Asn Glu Leu Asn Arg Met Ile Gln Arg Leu Thr Ala Glu Val
 325 330 335
 Glu Asn Ala Lys Cys Gln Asn Ser Lys Leu Glu Ala Ala Val Ala Gln
 340 345 350
 Ser Glu Gln Gln Gly Glu Ala Ala Leu Ser Asp Ala Arg Cys Lys Leu
 355 360 365
 Ala Glu Leu Glu Gly Ala Leu Gln Lys Ala Lys Gln Asp Met Ala Cys
 370 375 380
 Leu Ile Arg Glu Tyr Gln Glu Val Met Asn Ser Lys Leu Gly Leu Asp
 385 390 395 400
 Ile Glu Ile Ala Thr Tyr Arg Arg Leu Leu Glu Gly Glu Glu Gln Arg
 405 410 415
 Leu Cys Glu Gly Ile Gly Ala Val Asn Val Cys Val Ser Ser Ser Arg
 420 425 430
 Gly Gly Val Val Cys Gly Asp Leu Cys Val Ser Gly Ser Arg Pro Val
 435 440 445
 Thr Gly Ser Val Cys Ser Ala Pro Cys Asn Gly Asn Val Ala Val Ser
 450 455 460
 Thr Gly Leu Cys Ala Pro Cys Gly Gln Leu Asn Thr Thr Cys Gly Gly
 465 470 475 480
 Gly Ser Cys Gly Val Gly Ser Cys Gly Ile Ser Ser Leu Gly Val Gly
 485 490 495
 Ser Cys Gly Ser Ser Cys Arg Lys Cys
 500 505
 <210> 446
 <211> 486
 <212> PRT
 <213> Homo sapiens

<400> 446
 Met Thr Cys Gly Ser Tyr Cys Gly Gly Arg Ala Phe Ser Cys Ile Ser
 1 5 10 15
 Ala Cys Gly Pro Arg Pro Gly Arg Cys Cys Ile Thr Ala Ala Pro Tyr
 20 25 30
 Arg Gly Ile Ser Cys Tyr Arg Gly Leu Thr Gly Gly Phe Gly Ser His
 35 40 45
 Ser Val Cys Gly Gly Phe Arg Ala Gly Ser Cys Gly Arg Ser Phe Gly
 50 55 60
 Tyr Arg Ser Gly Gly Val Cys Gly Pro Ser Pro Pro Cys Ile Thr Thr
 65 70 75 80
 Val Ser Val Asn Glu Ser Leu Leu Thr Pro Leu Asn Leu Glu Ile Asp
 85 90 95
 Pro Asn Ala Gln Cys Val Lys Gln Glu Lys Glu Gln Ile Lys Ser
 100 105 110
 Leu Asn Ser Arg Phe Ala Ala Phe Ile Asp Lys Val Arg Phe Leu Glu
 115 120 125
 Gln Gln Asn Lys Leu Leu Glu Thr Lys Leu Gln Phe Tyr Gln Asn Arg
 130 135 140
 Glu Cys Cys Gln Ser Asn Leu Glu Pro Leu Phe Glu Gly Tyr Ile Glu
 145 150 155 160
 Thr Leu Arg Arg Glu Ala Glu Cys Val Glu Ala Asp Ser Gly Arg Leu
 165 170 175

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Ala	Ser	Glu	Leu	Asn	His	Val	Gln	Glu	Val	Leu	Glu	Gly	Tyr	Lys	Lys	
			180					185					190			
Lys	Tyr	Glu	Glu	Glu	Val	Ser	Leu	Arg	Ala	Thr	Ala	Glu	Asn	Glu	Phe	
		195					200					205				
Val	Ala	Leu	Lys	Lys	Asp	Val	Asp	Cys	Ala	Tyr	Leu	Arg	Lys	Ser	Asp	
		210				215					220					
Leu	Glu	Ala	Asn	Val	Glu	Ala	Leu	Ile	Gln	Glu	Ile	Asp	Phe	Leu	Arg	
		225			230					235					240	
Arg	Leu	Tyr	Glu	Glu	Ile	Arg	Val	Leu	Gln	Ser	His	Ile	Ser	Asp		
			245					250					255			
Thr	Ser	Val	Val	Val	Lys	Leu	Asp	Asn	Ser	Arg	Asp	Leu	Asn	Met	Asp	
			260					265					270			
Cys	Ile	Ile	Ala	Glu	Ile	Lys	Ala	Gln	Tyr	Asp	Asp	Ile	Val	Thr	Arg	
		275					280					285				
Ser	Arg	Ala	Glu	Ala	Glu	Ser	Trp	Tyr	Arg	Ser	Lys	Cys	Glu	Glu	Met	
		290				295					300					
Lys	Ala	Thr	Val	Ile	Arg	His	Gly	Glu	Thr	Leu	Arg	Arg	Thr	Lys	Glu	
		305			310					315					320	
Glu	Ile	Asn	Glu	Leu	Asn	Arg	Met	Ile	Gln	Arg	Leu	Thr	Ala	Glu	Val	
			325						330					335		
Glu	Asn	Ala	Lys	Cys	Gln	Asn	Ser	Lys	Leu	Glu	Ala	Ala	Val	Ala	Gln	
		340						345					350			
Ser	Glu	Gln	Gln	Gly	Glu	Ala	Ala	Leu	Ser	Asp	Ala	Arg	Cys	Lys	Leu	
		355					360					365				
Ala	Glu	Leu	Glu	Gly	Ala	Leu	Gln	Lys	Ala	Lys	Gln	Asp	Met	Ala	Cys	
		370				375					380					
Leu	Ile	Arg	Glu	Tyr	Gln	Glu	Val	Met	Asn	Ser	Lys	Leu	Gly	Leu	Asp	
		385			390					395					400	
Ile	Glu	Ile	Ala	Thr	Tyr	Arg	Arg	Leu	Leu	Glu	Gly	Glu	Glu	Gln	Arg	
			405					410						415		
Leu	Cys	Glu	Gly	Val	Gly	Ser	Val	Asn	Val	Cys	Val	Ser	Ser	Ser	Arg	
		420						425					430			
Gly	Gly	Val	Val	Cys	Gly	Asp	Leu	Cys	Ala	Ser	Thr	Thr	Ala	Pro	Val	
		435					440					445				
Val	Ser	Thr	Arg	Val	Ser	Ser	Val	Pro	Ser	Asn	Ser	Asn	Val	Val	Val	
		450				455					460					
Gly	Thr	Thr	Asn	Ala	Cys	Ala	Pro	Ser	Ala	Arg	Val	Gly	Val	Cys	Gly	
		465			470					475					480	
Gly	Ser	Cys	Lys	Arg	Cys											
				485												

<210> 447

<211> 493

<212> PRT

<213> Homo sapiens

<400>	447															
Met	Thr	Cys	Gly	Phe	Asn	Ser	Ile	Gly	Cys	Gly	Phe	Arg	Pro	Gly	Asn	
1			5						10					15		
Phe	Ser	Cys	Val	Ser	Ala	Cys	Gly	Pro	Arg	Pro	Ser	Arg	Cys	Cys	Ile	
			20					25					30			
Thr	Ala	Ala	Pro	Tyr	Arg	Gly	Ile	Ser	Cys	Tyr	Arg	Gly	Leu	Thr	Gly	
		35					40					45				
Gly	Phe	Gly	Ser	His	Ser	Val	Cys	Gly	Gly	Phe	Arg	Ala	Gly	Ser	Cys	
		50				55					60					
Gly	Arg	Ser	Phe	Gly	Tyr	Arg	Ser	Gly	Gly	Val	Cys	Gly	Pro	Ser	Pro	
		65			70					75					80	
Pro	Cys	Ile	Thr	Thr	Val	Ser	Val	Asn	Glu	Ser	Leu	Leu	Thr	Pro	Leu	
			85						90					95		
Asn	Leu	Glu	Ile	Asp	Pro	Asn	Ala	Gln	Cys	Val	Lys	Gln	Glu	Glu	Lys	
			100					105						110		

Glu	Gln	Ile	Lys	Ser	Leu	Asn	Ser	Arg	Phe	Ala	Ala	Phe	Ile	Asp	Lys
		115					120					125			
Val	Arg	Phe	Leu	Glu	Gln	Gln	Asn	Lys	Leu	Leu	Glu	Thr	Lys	Leu	Gln
		130				135					140				
Phe	Tyr	Gln	Asn	Cys	Glu	Cys	Cys	Gln	Ser	Asn	Leu	Glu	Pro	Leu	Phe
145					150					155					160
Ala	Gly	Tyr	Ile	Glu	Thr	Leu	Arg	Arg	Glu	Ala	Glu	Cys	Val	Glu	Ala
				165					170					175	
Asp	Ser	Gly	Arg	Leu	Ala	Ser	Glu	Leu	Asn	His	Val	Gln	Glu	Val	Leu
			180					185					190		
Glu	Gly	Tyr	Lys	Lys	Lys	Tyr	Glu	Glu	Glu	Val	Ala	Leu	Arg	Ala	Thr
		195					200					205			
Ala	Glu	Asn	Glu	Phe	Val	Ala	Leu	Lys	Lys	Asp	Val	Asp	Cys	Ala	Tyr
		210				215					220				
Leu	Arg	Lys	Ser	Asp	Leu	Glu	Ala	Asn	Val	Glu	Ala	Leu	Ile	Gln	Glu
225					230					235					240
Ile	Asp	Phe	Leu	Arg	Arg	Leu	Tyr	Glu	Glu	Glu	Ile	Arg	Ile	Leu	Gln
				245					250					255	
Ser	His	Ile	Ser	Asp	Thr	Ser	Val	Val	Val	Lys	Leu	Asp	Asn	Ser	Arg
			260					265					270		
Asp	Leu	Asn	Met	Asp	Cys	Ile	Val	Ala	Glu	Ile	Lys	Ala	Gln	Tyr	Asp
		275					280					285			
Asp	Ile	Ala	Thr	Arg	Ser	Arg	Ala	Glu	Ala	Glu	Ser	Trp	Tyr	Arg	Ser
		290				295					300				
Lys	Cys	Glu	Glu	Met	Lys	Ala	Thr	Val	Ile	Arg	His	Gly	Glu	Thr	Leu
305					310					315					320
Arg	Arg	Thr	Lys	Glu	Glu	Ile	Asn	Glu	Leu	Asn	Arg	Met	Ile	Gln	Arg
				325					330					335	
Leu	Thr	Ala	Glu	Val	Glu	Asn	Ala	Lys	Cys	Gln	Asn	Ser	Lys	Leu	Glu
			340					345					350		
Ala	Ala	Val	Ala	Gln	Ser	Glu	Gln	Gln	Gly	Glu	Ala	Ala	Leu	Ser	Asp
		355					360					365			
Ala	Arg	Cys	Lys	Leu	Ala	Glu	Leu	Glu	Gly	Ala	Leu	Gln	Lys	Ala	Lys
		370				375					380				
Gln	Asp	Met	Ala	Cys	Leu	Ile	Arg	Glu	Tyr	Gln	Glu	Val	Met	Asn	Ser
385					390					395					400
Lys	Leu	Gly	Leu	Asp	Ile	Glu	Ile	Ala	Thr	Tyr	Arg	Arg	Leu	Leu	Glu
				405					410					415	
Gly	Glu	Glu	Gln	Arg	Leu	Cys	Glu	Gly	Val	Glu	Ala	Val	Asn	Val	Cys
			420					425					430		
Val	Ser	Ser	Ser	Arg	Gly	Gly	Val	Val	Cys	Gly	Asp	Leu	Cys	Val	Ser
		435					440					445			
Gly	Ser	Arg	Pro</												

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<400>      448
Met Ala Ser Gln Ser Cys His Ile Ser Ser Gly Cys Gly Val Lys Asn
1              5              10              15
Phe Ser Ser Arg Ser Ala Thr Val Pro Lys Pro Gly Tyr His Ser Cys
              20              25              30
Val Ser Ala Met Ala His His Gly Val Ser Pro Gly Gly Leu Gly Ser
              35              40              45

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Arg Arg Leu Gly Gly Phe Gly Ser Gln Ser Leu Cys Thr Val Gly Ser
 50 55 60
 Pro Arg Ile Ala Val Ser Cys Arg Trp Pro Leu His Ser Arg Gly Arg
 65 70 75 80
 Phe Gly Tyr Trp Ala Gly Gly Leu Cys Arg Pro Ser Pro Pro Arg Ile
 85 90 95
 Thr Ser Val Thr Ile Asn Glu Ser Leu Leu Met Pro Leu Asn Leu Glu
 100 105 110
 Ile Asp Pro Asn Ala Gln Cys Val Lys His Glu Glu Lys Glu His Ile
 115 120 125
 Arg Cys Leu Asn Lys Phe Ala Ala Phe Ile Asp Lys Val Gly Leu
 130 135 140

<210> 449

<211> 507

<212> PRT

<213> Homo sapiens

<400> 449

Met Ser Cys Arg Ser Tyr Arg Ile Ser Ser Gly Cys Gly Val Thr Arg
 1 5 10 15
 Asn Phe Ser Ser Cys Ser Ala Val Ala Pro Lys Thr Gly Asn Arg Cys
 20 25 30
 Cys Ile Ser Ala Ala Pro Tyr Arg Gly Val Ser Cys Tyr Arg Gly Leu
 35 40 45
 Thr Gly Phe Gly Ser Arg Ser Leu Cys Asn Leu Gly Ser Cys Gly Pro
 50 55 60
 Arg Ile Ala Val Gly Gly Phe Arg Ala Gly Ser Cys Gly Arg Ser Phe
 65 70 75 80
 Gly Tyr Arg Ser Gly Gly Val Cys Gly Pro Ser Pro Pro Cys Ile Thr
 85 90 95
 Thr Val Ser Val Asn Glu Ser Leu Leu Thr Pro Leu Asn Leu Glu Ile
 100 105 110
 Asp Pro Asn Ala Gln Cys Val Lys Gln Glu Glu Lys Glu Gln Ile Lys
 115 120 125
 Ser Leu Asn Ser Arg Phe Ala Ala Phe Ile Asp Lys Val Arg Phe Leu
 130 135 140
 Glu Gln Gln Asn Lys Leu Leu Glu Thr Lys Trp Gln Phe Tyr Gln Asn
 145 150 155 160
 Gln Arg Cys Cys Glu Ser Asn Leu Glu Pro Leu Phe Ser Gly Tyr Ile
 165 170 175
 Glu Thr Leu Arg Arg Glu Ala Glu Cys Val Glu Ala Asp Ser Gly Arg
 180 185 190
 Leu Ala Ser Glu Leu Asn His Val Gln Glu Val Leu Glu Gly Tyr Lys
 195 200 205
 Lys Lys Tyr Glu Glu Glu Val Ala Leu Arg Ala Thr Ala Glu Asn Glu
 210 215 220
 Phe Val Val Leu Lys Lys Asp Val Asp Cys Ala Tyr Leu Arg Lys Ser
 225 230 235 240
 Asp Leu Glu Ala Asn Val Glu Ala Leu Val Glu Glu Ser Ser Phe Leu
 245 250 255
 Arg Arg Leu Tyr Glu Glu Glu Ile Arg Val Leu Gln Ala His Ile Ser
 260 265 270
 Asp Thr Ser Val Ile Val Lys Met Asp Asn Ser Arg Asp Leu Asn Met
 275 280 285
 Asp Cys Ile Ile Ala Glu Ile Lys Ala Gln Tyr Asp Asp Val Ala Ser
 290 295 300
 Arg Ser Arg Ala Glu Ala Glu Ser Trp Tyr Arg Ser Lys Cys Glu Glu
 305 310 315 320
 Met Lys Ala Thr Val Ile Arg His Gly Glu Thr Leu Arg Arg Thr Lys
 325 330 335

Glu Glu Ile Asn Glu Leu Asn Arg Met Ile Gln Arg Leu Thr Ala Glu
 340 345 350
 Ile Glu Asn Ala Lys Cys Gln Arg Ala Lys Leu Glu Ala Ala Val Ala
 355 360 365
 Glu Ala Glu Gln Gln Gly Glu Ala Ala Leu Ser Asp Ala Arg Cys Lys
 370 375 380
 Leu Ala Glu Leu Glu Gly Ala Leu Gln Lys Ala Lys Gln Asp Met Ala
 385 390 395 400
 Cys Leu Leu Lys Glu Tyr Gln Glu Val Met Asn Ser Lys Leu Gly Leu
 405 410 415
 Asp Ile Glu Ile Ala Thr Tyr Arg Arg Leu Leu Glu Gly Glu Glu His
 420 425 430
 Arg Leu Cys Glu Gly Val Gly Ser Val Asn Val Cys Val Ser Ser Ser
 435 440 445
 Arg Gly Gly Val Ser Cys Gly Gly Leu Ser Tyr Ser Thr Thr Pro Gly
 450 455 460
 Arg Gln Ile Thr Ser Gly Pro Ser Ala Ile Gly Gly Ser Ile Thr Val
 465 470 475 480
 Val Ala Pro Asp Ser Cys Ala Pro Cys Gln Pro Arg Ser Ser Ser Phe
 485 490 495
 Ser Cys Gly Ser Ser Arg Ser Val Arg Phe Ala
 500 505

<210> 450

<211> 600

<212> PRT

<213> Homo sapiens

<400> 450

Met Ser Cys Arg Ser Tyr Arg Val Ser Ser Gly His Arg Val Gly Asn
 1 5 10 15
 Phe Ser Ser Cys Ser Ala Met Thr Pro Gln Asn Leu Asn Arg Phe Arg
 20 25 30
 Ala Asn Ser Val Ser Cys Trp Ser Gly Pro Gly Phe Arg Gly Leu Gly
 35 40 45
 Ser Phe Gly Ser Arg Ser Val Ile Thr Phe Gly Ser Tyr Ser Pro Arg
 50 55 60
 Ile Ala Ala Val Gly Ser Arg Pro Ile His Cys Gly Val Arg Phe Gly
 65 70 75 80
 Ala Gly Cys Gly Met Gly Phe Gly Asp Gly Arg Gly Val Gly Leu Gly
 85 90 95
 Pro Arg Ala Asp Ser Cys Val Gly Leu Gly Phe Gly Ala Gly Ser Gly
 100 105 110
 Ile Gly Tyr Gly Phe Gly Gly Pro Gly Phe Gly Tyr Arg Val Gly Gly
 115 120 125
 Val Gly Val Pro Ala Ala Pro Ser Ile Thr Ala Val Thr Val Asn Lys
 130 135 140
 Ser Leu Leu Thr Pro Leu Asn Leu Glu Ile Asp Pro Asn Ala Gln Arg
 145 150 155 160
 Val Lys Lys Asp Glu Lys Glu Gln Ile Lys Thr Leu Asn Asn Lys Phe
 165 170 175
 Ala Ser Phe Ile Asp Lys Val Arg Phe Leu Glu Gln Gln Asn Lys Leu
 180 185 190
 Leu Glu Thr Lys Trp Ser Phe Leu Gln Glu Gln Lys Cys Ile Arg Ser
 195 200 205
 Asn Leu Glu Pro Leu Phe Glu Ser Tyr Ile Thr Asn Leu Arg Arg Gln
 210 215 220
 Leu Glu Val Leu Val Ser Asp Gln Ala Arg Leu Gln Ala Glu Arg Asn
 225 230 235 240
 His Leu Gln Asp Val Leu Glu Gly Phe Lys Lys Lys Tyr Glu Glu Glu
 245 250 255

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Val	Val	Cys	Arg	Ala	Asn	Ala	Glu	Asn	Glu	Phe	Val	Ala	Leu	Lys	Lys
			260					265					270		
Asp	Val	Asp	Ala	Ala	Phe	Met	Asn	Lys	Ser	Asp	Leu	Glu	Ala	Asn	Val
		275					280					285			
Asp	Thr	Leu	Thr	Gln	Glu	Ile	Asp	Phe	Leu	Lys	Thr	Leu	Tyr	Met	Glu
		290				295					300				
Glu	Ile	Gln	Leu	Leu	Gln	Ser	His	Ile	Ser	Glu	Thr	Ser	Val	Ile	Val
					310					315					320
Lys	Met	Asp	Asn	Ser	Arg	Asp	Leu	Asn	Leu	Asp	Gly	Ile	Ile	Ala	Glu
			325					330						335	
Val	Lys	Ala	Gln	Tyr	Glu	Glu	Val	Ala	Arg	Arg	Ser	Arg	Ala	Asp	Ala
			340					345					350		
Glu	Ala	Trp	Tyr	Gln	Thr	Lys	Tyr	Glu	Glu	Met	Gln	Val	Thr	Ala	Gly
		355					360					365			
Gln	His	Cys	Asp	Asn	Leu	Arg	Asn	Ile	Arg	Asn	Glu	Ile	Asn	Glu	Leu
		370				375					380				
Thr	Arg	Leu	Ile	Gln	Arg	Leu	Lys	Ala	Glu	Ile	Glu	His	Ala	Lys	Ala
				390					395						400
Gln	Arg	Ala	Lys	Leu	Glu	Ala	Ala	Val	Ala	Glu	Ala	Glu	Gln	Gln	Gly
				405					410					415	
Glu	Ala	Thr	Leu	Ser	Asp	Ala	Lys	Cys	Lys	Leu	Ala	Asp	Leu	Glu	Cys
			420					425					430		
Ala	Leu	Gln	Gln	Ala	Lys	Gln	Asp	Met	Ala	Arg	Gln	Leu	Cys	Glu	Tyr
		435					440					445			
Gln	Glu	Leu	Met	Asn	Ala	Lys	Leu	Gly	Leu	Asp	Ile	Glu	Ile	Ala	Thr
		450				455					460				
Tyr	Arg	Arg	Leu	Leu	Glu	Gly	Glu	Glu	Ser	Arg	Leu	Cys	Glu	Gly	Val
				470						475					480
Gly	Pro	Val	Asn	Ile	Ser	Val	Ser	Ser	Ser	Arg	Gly	Gly	Leu	Val	Cys
			485					490						495	
Gly	Pro	Glu	Pro	Leu	Val	Ala	Gly	Ser	Thr	Leu	Ser	Arg	Gly	Gly	Val
			500					505					510		
Thr	Phe	Ser	Gly	Ser	Ser	Ser	Val	Cys	Ala	Thr	Ser	Gly	Val	Leu	Ala
		515					520					525			
Ser	Cys	Gly	Pro	Ser	Leu	Gly	Gly	Ala	Arg	Val	Ala	Pro	Ala	Thr	Gly
		530				535					540				
Asp	Leu	Leu	Ser	Thr	Gly	Thr	Arg	Ser	Gly	Ser	Met	Leu	Ile	Ser	Glu
				550						555					560
Ala	Cys	Val	Pro	Ser	Val	Pro	Cys	Pro	Leu	Pro	Thr	Gln	Gly	Gly	Phe
				565					570					575	
Ser	Ser	Cys	Ser	Gly	Gly	Arg	Ser	Ser	Ser	Val	Arg	Phe	Val	Ser	Thr
			580					585					590		
Thr	Thr	Ser	Cys	Arg	Thr	Lys	Tyr								
		595					600								

<210> 451

<211> 513

<212> PRT

<213> Homo sapiens

<400> 451

Met	Ser	Tyr	His	Ser	Phe	Gln	Pro	Gly	Ser	Arg	Cys	Gly	Ser	Gln	Ser
1			5						10					15	
Phe	Ser	Ser	Tyr	Ser	Ala	Val	Met	Pro	Arg	Met	Val	Thr	His	Tyr	Ala
			20					25				30			
Val	Ser	Lys	Gly	Pro	Cys	Arg	Pro	Gly	Gly	Gly	Arg	Gly	Leu	Arg	Ala
		35					40					45			
Leu	Gly	Cys	Leu	Gly	Ser	Arg	Ser	Leu	Cys	Asn	Val	Gly	Phe	Gly	Arg
	50					55					60				
Pro	Arg	Val	Ala	Ser	Arg	Cys	Gly	Gly	Thr	Leu	Pro	Gly	Phe	Gly	Tyr
65					70					75					80

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Arg	Leu	Gly	Ala	Thr	Cys	Gly	Pro	Ser	Ala	Cys	Ile	Thr	Pro	Val	Thr
				85					90					95	
Ile	Asn	Glu	Ser	Leu	Leu	Val	Pro	Leu	Ala	Leu	Glu	Ile	Asp	Pro	Thr
			100					105					110		
Val	Gln	Arg	Val	Lys	Arg	Asp	Glu	Lys	Glu	Gln	Ile	Lys	Cys	Leu	Asn
		115					120					125			
Asn	Arg	Phe	Ala	Ser	Phe	Ile	Asn	Lys	Val	Arg	Phe	Leu	Glu	Gln	Lys
		130				135					140				
Asn	Lys	Leu	Leu	Glu	Thr	Lys	Trp	Asn	Phe	Met	Gln	Gln	Gln	Arg	Cys
145					150					155					160
Cys	Gln	Thr	Asn	Ile	Glu	Pro	Ile	Phe	Glu	Gly	Tyr	Ile	Ser	Ala	Leu
				165					170						175
Arg	Arg	Gln	Leu	Asp	Cys	Val	Ser	Gly	Asp	Arg	Val	Arg	Leu	Glu	Ser
			180					185					190		
Glu	Leu	Cys	Ser	Leu	Gln	Ala	Ala	Leu	Glu	Gly	Tyr	Lys	Lys	Lys	Tyr
		195					200					205			
Glu	Glu	Glu	Leu	Ser	Leu	Arg	Pro	Cys	Val	Glu	Asn	Glu	Phe	Val	Ala
		210				215					220				
Leu	Lys	Lys	Asp	Val	Asp	Thr	Ala	Phe	Leu	Met	Lys	Ala	Asp	Leu	Glu
225					230					235					240
Thr	Asn	Ala	Glu	Ala	Leu	Val	Gln	Glu	Ile	Asp	Phe	Leu	Lys	Ser	Leu
				245					250						255
Tyr	Glu	Glu	Glu	Ile	Cys	Leu	Leu	Gln	Ser	Gln	Ile	Ser	Glu	Thr	Ser
			260					265					270		
Val	Ile	Val	Lys	Met	Asp	Asn	Ser	Arg	Glu	Leu	Asp	Val	Asp	Gly	Ile
		275					280					285			
Ile	Ala	Glu	Ile	Lys	Ala	Gln	Tyr	Asp	Asp	Ile	Ala	Ser	Arg	Ser	Lys
		290				295					300				
Ala	Glu	Ala	Glu	Ala	Trp	Tyr	Gln	Cys	Arg	Tyr	Glu	Glu	Leu	Arg	Val
305					310					315					320
Thr	Ala	Gly	Asn	His	Cys	Asp	Asn	Leu	Arg	Asn	Arg	Lys	Asn	Glu	Ile
				325					330					335	
Leu	Glu	Met	Asn	Lys	Leu	Ile	Gln	Arg	Leu	Gln	Gln	Glu	Thr	Glu	Asn
			340					345					350		
Val	Lys	Ala	Gln	Arg	Cys	Lys	Leu	Glu	Gly	Ala	Ile	Ala	Glu	Ala	Glu
		355					360					365			
Gln	Gln	Gly	Glu	Ala	Ala	Leu	Asn	Asp	Ala	Lys	Cys	Lys	Leu	Ala	Gly
		370				375					380				
Leu	Glu	Glu	Ala	Leu	Gln	Lys	Ala	Lys	Gln	Asp	Met	Ala	Cys	Leu	Leu
385					390					395					400
Lys	Glu	Tyr	Gln	Glu	Val	Met	Asn	Ser	Lys	Leu	Gly	Leu	Asp	Ile	Glu
				405					410					415	
Ile	Ala	Thr	Tyr	Arg	Arg	Leu	Leu	Glu	Gly	Glu	Glu	His	Arg	Leu	Cys
			420					425					430		
Glu	Gly	Ile	Gly	Pro	Val	Asn	Ile	Ser	Val	Ser	Ser	Ser	Lys	Gly	Ala
		435					440					445			
Phe	Leu	Tyr	Glu	Pro	Cys	Gly	Val	Ser	Thr	Pro	Val	Leu	Ser	Thr	Gly
		450				455					460				
Val	Leu	Arg	Ser	Asn	Gly	Gly	Cys	Ser	Ile	Val	Gly	Thr	Gly	Glu	Leu
465					470					475					480
Tyr	Val	Pro	Cys	Glu	Pro	Gln	Gly	Leu	Leu	Ser	Cys	Gly	Ser	Gly	Arg
				485					490					495	
Lys	Ser	Ser	Met	Thr	Leu	Gly	Ala	Gly	Gly	Ser	Ser	Pro	Ser	His	Lys
			500					505					510		

His

<210> 452

<211> 85

<212> PRT

<213> Homo sapiens

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<400> 452

Met Asp Ala Val Tyr Met Asn Lys Val Gly Leu Glu Ala Lys Val Asp
 1 5 10 15
 Ala Leu Met Glu Glu Thr Asn Phe Leu Ser Thr Phe Tyr Lys Ala Val
 20 25 30
 Arg Val Pro Gly Ala Pro Ser Asn Arg Gly Ala Gly Gly Trp Val Leu
 35 40 45
 Glu Pro Gln Leu Gly Thr Glu Pro Val Gly Ser Phe Pro Gly Leu Leu
 50 55 60
 Ser Ala Pro Tyr Pro Thr Cys Val Leu Gln Gly Arg Cys His Phe Pro
 65 70 75 80
 Tyr His Arg Arg Lys
 85

<210> 453

<211> 564

<212> PRT

<213> Homo sapiens

<400> 453

Met Ala Ser Thr Ser Thr Thr Ile Arg Ser His Ser Ser Ser Arg Arg
 1 5 10 15
 Gly Phe Ser Ala Ser Ser Ala Arg Leu Pro Gly Val Ser Arg Ser Gly
 20 25 30
 Phe Ser Ser Ile Ser Val Ser Arg Ser Arg Gly Ser Gly Gly Leu Gly
 35 40 45
 Gly Ala Cys Gly Gly Ala Gly Phe Gly Ser Arg Ser Leu Tyr Gly Leu
 50 55 60
 Gly Gly Ser Lys Arg Ile Ser Ile Gly Gly Gly Ser Cys Ala Ile Ser
 65 70 75 80
 Gly Gly Tyr Gly Ser Arg Ala Gly Gly Ser Tyr Gly Phe Gly Gly Ala
 85 90 95
 Gly Ser Gly Phe Gly Phe Gly Gly Ala Gly Ile Gly Phe Gly Leu
 100 105 110
 Gly Gly Gly Ala Gly Leu Ala Gly Phe Gly Gly Pro Gly Phe Pro
 115 120 125
 Val Cys Pro Pro Gly Gly Ile Gln Glu Val Thr Val Asn Gln Ser Leu
 130 135 140
 Leu Thr Pro Leu Asn Leu Gln Ile Asp Pro Ala Ile Gln Arg Val Arg
 145 150 155 160
 Ala Glu Glu Arg Glu Gln Ile Lys Thr Leu Asn Asn Lys Phe Ala Ser
 165 170 175
 Phe Ile Asp Lys Val Arg Phe Leu Glu Gln Gln Asn Lys Val Leu Asp
 180 185 190
 Thr Lys Trp Thr Leu Leu Gln Glu Gln Gly Thr Lys Thr Val Arg Gln
 195 200 205
 Asn Leu Glu Pro Leu Phe Glu Gln Tyr Ile Asn Asn Leu Arg Arg Gln
 210 215 220
 Leu Asp Ser Ile Val Gly Glu Arg Gly Arg Leu Asp Ser Glu Leu Arg
 225 230 235 240
 Asn Met Gln Asp Leu Val Glu Asp Leu Lys Asn Lys Tyr Glu Asp Glu
 245 250 255
 Ile Asn Lys Arg Thr Ala Ala Glu Asn Glu Phe Val Thr Leu Lys Lys
 260 265 270
 Asp Val Asp Ala Ala Tyr Met Asn Lys Val Glu Leu Gln Ala Lys Ala
 275 280 285
 Asp Thr Leu Thr Asp Glu Ile Asn Phe Leu Arg Ala Leu Tyr Asp Ala
 290 295 300
 Glu Leu Ser Gln Met Gln Thr His Ile Ser Asp Thr Ser Val Val Leu
 305 310 315 320
 Ser Met Asp Asn Asn Arg Asn Leu Asp Leu Asp Ser Ile Ile Ala Glu
 325 330 335

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Val Lys Ala Gln Tyr Glu Glu Ile Ala Gln Arg Ser Arg Ala Glu Ala
 340 345 350
 Glu Ser Trp Tyr Gln Thr Lys Tyr Glu Glu Leu Gln Val Thr Ala Gly
 355 360 365
 Arg His Gly Asp Asp Leu Arg Asn Thr Lys Gln Glu Ile Ala Glu Ile
 370 375 380
 Asn Arg Met Ile Gln Arg Leu Arg Ser Glu Ile Asp His Val Lys Lys
 385 390 395 400
 Gln Cys Ala Asn Leu Gln Ala Ala Ile Ala Asp Ala Glu Gln Arg Gly
 405 410 415
 Glu Met Ala Leu Lys Asp Ala Lys Asn Lys Leu Glu Gly Leu Glu Asp
 420 425 430
 Ala Leu Gln Lys Ala Lys Gln Asp Leu Ala Arg Leu Leu Lys Glu Tyr
 435 440 445
 Gln Glu Leu Met Asn Val Lys Leu Ala Leu Asp Val Glu Ile Ala Thr
 450 455 460
 Tyr Arg Lys Leu Leu Glu Gly Glu Glu Cys Arg Leu Asn Gly Glu Gly
 465 470 475 480
 Val Gly Gln Val Asn Ile Ser Val Val Gln Ser Thr Val Ser Ser Gly
 485 490 495
 Tyr Gly Gly Ala Ser Gly Val Gly Ser Gly Leu Gly Leu Gly Gly Gly
 500 505 510
 Ser Ser Tyr Ser Tyr Gly Ser Gly Leu Gly Val Gly Gly Gly Phe Ser
 515 520 525
 Ser Ser Ser Gly Arg Ala Thr Gly Gly Gly Leu Ser Ser Val Gly Gly
 530 535 540
 Gly Ser Ser Thr Ile Lys Tyr Thr Thr Thr Ser Ser Ser Arg Lys
 545 550 555 560
 Ser Tyr Lys His

<210> 454

<211> 564

<212> PRT

<213> Homo sapiens

<400> 454

Met Ala Ser Thr Ser Thr Thr Ile Arg Ser His Ser Ser Ser Arg Arg
 1 5 10 15
 Gly Phe Ser Ala Asn Ser Ala Arg Leu Pro Gly Val Ser Arg Ser Gly
 20 25 30
 Phe Ser Ser Ile Ser Val Ser Arg Ser Arg Gly Ser Gly Gly Leu Gly
 35 40 45
 Gly Ala Cys Gly Gly Ala Gly Phe Gly Ser Arg Ser Leu Tyr Gly Leu
 50 55 60
 Gly Gly Ser Lys Arg Ile Ser Ile Gly Gly Gly Ser Cys Ala Ile Ser
 65 70 75 80
 Gly Gly Tyr Gly Ser Arg Ala Arg Gly Ser Tyr Gly Phe Gly Gly Ala
 85 90 95
 Gly Ser Gly Phe Gly Phe Gly Gly Ala Gly Ile Gly Phe Asp Leu
 100 105 110
 Gly Gly Gly Ala Gly Leu Ala Gly Phe Gly Gly Pro Gly Phe Pro
 115 120 125
 Val Cys Pro Pro Gly Gly Ile Gln Glu Val Thr Val Asn Gln Ser Leu
 130 135 140
 Leu Thr Pro Leu Asn Leu Gln Ile Asp Pro Ala Ile Gln Arg Val Arg
 145 150 155 160
 Ala Glu Glu Arg Glu Gln Ile Lys Thr Leu Asn Asn Lys Phe Ala Ser
 165 170 175
 Phe Ile Asp Lys Val Arg Phe Leu Glu Gln Gln Asn Lys Val Leu Asp
 180 185 190

Thr Lys Trp Thr Leu Leu Gln Glu Gln Gly Thr Lys Thr Val Arg Gln
 195 200 205
 Asn Leu Glu Pro Leu Phe Glu Gln Tyr Ile Asn Asn Leu Arg Arg Gln
 210 215 220
 Leu Asp Asn Ile Val Gly Glu Arg Gly Arg Leu Asp Ser Glu Leu Arg
 225 230 235 240
 Asn Met Gln Asp Leu Val Glu Asp Leu Lys Asn Lys Tyr Glu Asp Glu
 245 250 255
 Ile Asn Lys Arg Thr Ala Ala Glu Asn Glu Phe Val Thr Leu Lys Lys
 260 265 270
 Asp Val Asp Ala Ala Tyr Met Asn Lys Val Glu Leu Gln Ala Lys Ala
 275 280 285
 Asp Thr Leu Thr Asp Glu Ile Asn Phe Leu Arg Ala Leu Tyr Asp Ala
 290 295 300
 Glu Leu Ser Gln Met Gln Thr His Ile Ser Asp Thr Ser Val Val Leu
 305 310 315 320
 Ser Met Asp Asn Asn Arg Asn Leu Asp Leu Asp Ser Ile Ile Ala Glu
 325 330 335
 Val Lys Ala Gln Tyr Glu Glu Ile Ala Gln Arg Ser Arg Ala Glu Ala
 340 345 350
 Glu Ser Trp Tyr Gln Thr Lys Tyr Glu Glu Leu Gln Val Thr Ala Gly
 355 360 365
 Arg His Gly Asp Asp Leu Arg Asn Thr Lys Gln Glu Ile Ala Glu Ile
 370 375 380
 Asn Arg Met Ile Gln Arg Leu Arg Ser Glu Ile Asp His Val Lys Lys
 385 390 395 400
 Gln Cys Ala Ser Leu Gln Ala Ala Ile Ala Asp Ala Glu Gln Arg Gly
 405 410 415
 Glu Met Ala Leu Lys Asp Ala Lys Asn Lys Leu Glu Gly Leu Glu Asp
 420 425 430
 Ala Leu Gln Lys Ala Lys Gln Asp Leu Ala Arg Leu Leu Lys Glu Tyr
 435 440 445
 Gln Glu Leu Met Asn Val Lys Leu Ala Leu Asp Val Glu Ile Ala Thr
 450 455 460
 Tyr Arg Lys Leu Leu Glu Gly Glu Glu Cys Arg Leu Asn Gly Glu Gly
 465 470 475 480
 Ile Gly Gln Val Asn Val Ser Val Val Gln Ser Thr Ile Ser Ser Gly
 485 490 495
 Tyr Gly Gly Ala Ser Gly Val Gly Ser Gly Leu Gly Leu Gly Gly Gly
 500 505 510
 Ser Ser Tyr Ser Tyr Gly Ser Gly Leu Gly Ile Gly Gly Gly Phe Ser
 515 520 525
 Ser Ser Ser Gly Arg Ala Ile Gly Gly Gly Leu Ser Ser Val Gly Gly
 530 535 540
 Gly Ser Ser Thr Ile Lys Tyr Thr Thr Thr Ser Ser Ser Arg Lys
 545 550 555 560
 Ser Tyr Lys His

<210> 455

<211> 564

<212> PRT

<213> Homo sapiens

<400> 455

Met Ala Ser Thr Ser Thr Thr Ile Arg Ser His Ser Ser Ser Arg Arg
 1 5 10 15
 Gly Phe Ser Ala Asn Ser Ala Arg Leu Pro Gly Val Ser Arg Ser Gly
 20 25 30
 Phe Ser Ser Ile Ser Val Ser Arg Ser Arg Gly Ser Gly Gly Leu Gly
 35 40 45

Gly Ala Cys Gly Gly Ala Gly Phe Gly Ser Arg Ser Leu Tyr Gly Leu
 50 55 60
 Gly Gly Ser Lys Arg Ile Ser Ile Gly Gly Gly Ser Cys Ala Ile Ser
 65 70 75 80
 Gly Gly Tyr Gly Ser Arg Ala Arg Ala Ser Tyr Gly Phe Gly Gly Ala
 85 90 95
 Gly Ser Gly Phe Gly Phe Gly Gly Gly Ala Gly Ile Gly Phe Asp Leu
 100 105 110
 Gly Gly Gly Ala Gly Leu Ala Gly Gly Phe Gly Gly Pro Gly Phe Pro
 115 120 125
 Val Cys Pro Pro Gly Gly Ile Gln Glu Val Thr Val Asn Gln Ser Leu
 130 135 140
 Leu Thr Pro Leu Asn Leu Gln Ile Asp Pro Ala Ile Gln Arg Val Arg
 145 150 155 160
 Ala Glu Glu Arg Glu Gln Ile Lys Thr Leu Asn Asn Lys Phe Ala Ser
 165 170 175
 Phe Ile Asp Lys Val Arg Phe Leu Glu Gln Gln Asn Lys Val Leu Glu
 180 185 190
 Thr Lys Trp Thr Leu Leu Gln Glu Gln Gly Thr Lys Thr Val Arg Gln
 195 200 205
 Asn Leu Glu Pro Leu Phe Glu Gln Tyr Ile Asn Asn Leu Arg Arg Gln
 210 215 220
 Leu Asp Ser Ile Val Gly Glu Arg Gly Arg Leu Asp Ser Glu Leu Arg
 225 230 235 240
 Gly Met Gln Asp Leu Val Glu Asp Phe Lys Asn Lys Tyr Glu Asp Glu
 245 250 255
 Ile Asn Lys Arg Thr Ala Ala Glu Asn Glu Phe Val Thr Leu Lys Lys
 260 265 270
 Asp Val Asp Ala Ala Tyr Met Asn Lys Val Glu Leu Gln Ala Lys Ala
 275 280 285
 Asp Thr Leu Thr Asp Glu Ile Asn Phe Leu Arg Ala Leu Tyr Asp Ala
 290 295 300
 Glu Leu Ser Gln Met Gln Thr His Ile Ser Asp Thr Ser Val Val Leu
 305 310 315 320
 Ser Met Asp Asn Asn Arg Asn Leu Asp Leu Asp Ser Ile Ile Ala Glu
 325 330 335
 Val Lys Ala Gln Tyr Glu Glu Ile Ala Gln Arg Ser Arg Ala Glu Ala
 340 345 350
 Glu Ser Trp Tyr Gln Thr Lys Tyr Glu Glu Leu Gln Val Thr Ala Gly
 355 360 365
 Arg His Gly Asp Asp Leu Arg Asn Thr Lys Gln Glu Ile Ala Glu Ile
 370 375 380
 Asn Arg Met Ile Gln Arg Leu Arg Ser Glu Ile Asp His Val Lys Lys
 385 390 395 400
 Gln Cys Ala Asn Leu Gln Ala Ala Ile Ala Asp Ala Glu Gln Arg Gly
 405 410 415
 Glu Met Ala Leu Lys Asp Ala Lys Asn Lys Leu Glu Gly Leu Glu Asp
 420 425 430
 Ala Leu Gln Lys Ala Lys Gln Asp Leu Ala Arg Leu Leu Lys Glu Tyr
 435 440 445
 Gln Glu Leu Met Asn Val Lys Leu Ala Leu Asp Val Glu Ile Ala Thr
 450 455 460
 Tyr Arg Lys Leu Leu Glu Gly Glu Glu Cys Arg Leu Asn Gly Glu Gly
 465 470 475 480
 Val Gly Gln Val Asn Ile Ser Val Val Gln Ser Thr Val Ser Ser Gly
 485 490 495
 Tyr Gly Gly Ala Ser Gly Val Gly Ser Gly Leu Gly Leu Gly Gly Gly
 500 505 510
 Ser Ser Tyr Ser Tyr Gly Ser Gly Leu Gly Val Gly Gly Gly Phe Ser
 515 520 525
 Ser Ser Ser Gly Arg Ala Ile Gly Gly Gly Leu Ser Ser Val Gly Gly
 530 535 540
 Gly Ser Ser Thr Ile Lys Tyr Thr Thr Thr Ser Ser Ser Arg Lys
 545 550 555 560
 Ser Tyr Lys His

<210> 456

<211> 564

<212> PRT

<213> Homo sapiens

<400> 456

Met	Ala	Ser	Thr	Ser	Thr	Thr	Ile	Arg	Ser	His	Ser	Ser	Ser	Arg	Arg
1				5					10					15	
Gly	Phe	Ser	Ala	Asn	Ser	Ala	Arg	Leu	Pro	Gly	Val	Ser	Arg	Ser	Gly
			20					25					30		
Phe	Ser	Ser	Val	Ser	Val	Ser	Arg	Ser	Arg	Gly	Ser	Gly	Gly	Leu	Gly
		35					40					45			
Gly	Ala	Cys	Gly	Gly	Ala	Gly	Phe	Gly	Ser	Arg	Ser	Leu	Tyr	Gly	Leu
	50					55					60				
Gly	Gly	Ser	Lys	Arg	Ile	Ser	Ile	Gly	Gly	Gly	Ser	Cys	Ala	Ile	Ser
65				70						75				80	
Gly	Gly	Tyr	Gly	Ser	Arg	Ala	Gly	Gly	Ser	Tyr	Gly	Phe	Gly	Gly	Ala
				85					90					95	
Gly	Ser	Gly	Phe	Gly	Phe	Gly	Gly	Gly	Ala	Gly	Ile	Gly	Phe	Gly	Leu
			100					105					110		
Gly	Gly	Gly	Ala	Gly	Leu	Ala	Gly	Gly	Phe	Gly	Gly	Pro	Gly	Phe	Pro
		115					120					125			
Val	Cys	Pro	Pro	Gly	Gly	Ile	Gln	Glu	Val	Thr	Val	Asn	Gln	Ser	Leu
	130					135					140				
Leu	Thr	Pro	Leu	Asn	Leu	Gln	Ile	Asp	Pro	Thr	Ile	Gln	Arg	Val	Arg
145				150						155				160	
Ala	Glu	Glu	Arg	Glu	Gln	Ile	Lys	Thr	Leu	Asn	Asn	Lys	Phe	Ala	Ser
				165					170					175	
Phe	Ile	Asp	Lys	Val	Arg	Phe	Leu	Glu	Gln	Gln	Asn	Lys	Val	Leu	Glu
			180					185					190		
Thr	Lys	Trp	Thr	Leu	Leu	Gln	Glu	Gln	Gly	Thr	Lys	Thr	Val	Arg	Gln
		195					200					205			
Asn	Leu	Glu	Pro	Leu	Phe	Glu	Gln	Tyr	Ile	Asn	Asn	Leu	Arg	Arg	Gln
	210					215					220				
Leu	Asp	Ser	Ile	Val	Gly	Glu	Arg	Gly	Arg	Leu	Asp	Ser	Glu	Leu	Arg
225				230						235				240	
Gly	Met	Gln	Asp	Leu	Val	Glu	Asp	Phe	Lys	Asn	Lys	Tyr	Glu	Asp	Glu
				245					250					255	
Ile	Asn	Lys	Arg	Thr	Ala	Ala	Glu	Asn	Glu	Phe	Val	Thr	Leu	Lys	Lys
			260					265					270		
Asp	Val	Asp	Ala	Ala	Tyr	Met	Asn	Lys	Val	Glu	Leu	Gln	Ala	Lys	Ala
		275					280					285			
Asp	Thr	Leu	Thr	Asp	Glu	Ile	Asn	Phe	Leu	Arg	Ala	Leu	Tyr	Asp	Ala
	290					295					300				
Glu	Leu	Ser	Gln	Met	Gln	Thr	His	Ile	Ser	Asp	Thr	Ser	Val	Val	Leu
305				310						315				320	
Ser	Met	Asp	Asn	Asn	Arg	Asn	Leu	Asp	Leu	Asp	Ser	Ile	Ile	Ala	Glu
			325						330					335	
Val	Lys	Ala	Gln	Tyr	Glu	Glu	Ile	Ala	Gln	Arg	Ser	Arg	Ala	Glu	Ala
			340					345					350		
Glu	Ser	Trp	Tyr	Gln	Thr	Lys	Tyr	Glu	Glu	Leu	Gln	Val	Thr	Ala	Gly
		355					360					365			
Arg	His	Gly	Asp	Asp	Leu	Arg	Asn	Thr	Lys	Gln	Glu	Ile	Ala	Glu	Ile
	370					375					380				
Asn	Arg	Met	Ile	Gln	Arg	Leu	Arg	Ser	Glu	Ile	Asp	His	Val	Lys	Lys
385				390						395				400	
Gln	Cys	Ala	Asn	Leu	Gln	Ala	Ala	Ile	Ala	Asp	Ala	Glu	Gln	Arg	Gly
			405					410					415		
Glu	Met	Ala	Leu	Lys	Asp	Ala	Lys	Asn	Lys	Leu	Glu	Gly	Leu	Glu	Asp
			420					425					430		

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Ala Leu Gln Lys Ala Lys Gln Asp Leu Ala Arg Leu Leu Lys Glu Tyr
 435 440 445
 Gln Glu Leu Met Asn Val Lys Leu Ala Leu Asp Val Glu Ile Ala Thr
 450 455 460
 Tyr Arg Lys Leu Leu Glu Gly Glu Glu Cys Arg Leu Asn Gly Glu Gly
 465 470 475 480
 Val Gly Gln Val Asn Ile Ser Val Val Gln Ser Thr Val Ser Ser Gly
 485 490 495
 Tyr Gly Gly Ala Ser Gly Val Gly Ser Gly Leu Gly Leu Gly Gly Gly
 500 505 510
 Ser Ser Tyr Ser Tyr Gly Ser Gly Leu Gly Val Gly Gly Gly Phe Ser
 515 520 525
 Ser Ser Ser Gly Arg Ala Ile Gly Gly Gly Leu Ser Ser Val Gly Gly
 530 535 540
 Gly Ser Ser Thr Ile Lys Tyr Thr Thr Thr Ser Ser Ser Arg Lys
 545 550 555 560
 Ser Tyr Lys His

<210> 457

<211> 590

<212> PRT

<213> Homo sapiens

<400> 457

Met Ser Arg Gln Ser Ser Val Ser Phe Arg Ser Gly Gly Ser Arg Ser
 1 5 10 15
 Phe Ser Thr Ala Ser Ala Ile Thr Pro Ser Val Ser Arg Thr Ser Phe
 20 25 30
 Thr Ser Val Ser Arg Ser Gly Gly Gly Gly Gly Gly Phe Gly Arg
 35 40 45
 Val Ser Leu Ala Gly Ala Cys Gly Val Gly Gly Tyr Gly Ser Arg Ser
 50 55 60
 Leu Tyr Asn Leu Gly Gly Ser Lys Arg Ile Ser Ile Ser Thr Arg Gly
 65 70 75 80
 Gly Ser Phe Arg Asn Arg Phe Gly Ala Gly Ala Gly Gly Gly Tyr Gly
 85 90 95
 Phe Gly Gly Gly Ala Gly Ser Gly Phe Gly Phe Gly Gly Gly Ala Gly
 100 105 110
 Gly Gly Phe Gly Leu Gly Gly Gly Ala Gly Phe Gly Gly Phe Gly
 115 120 125
 Gly Pro Gly Phe Pro Val Cys Pro Pro Gly Gly Ile Gln Glu Val Thr
 130 135 140
 Val Asn Gln Ser Leu Leu Thr Pro Leu Asn Leu Gln Ile Asp Pro Ser
 145 150 155 160
 Ile Gln Arg Val Arg Thr Glu Glu Arg Glu Gln Ile Lys Thr Leu Asn
 165 170 175
 Asn Lys Phe Ala Ser Phe Ile Asp Lys Val Arg Phe Leu Glu Gln Gln
 180 185 190
 Asn Lys Val Leu Asp Thr Lys Trp Thr Leu Leu Gln Glu Gln Gly Thr
 195 200 205
 Lys Thr Val Arg Gln Asn Leu Glu Pro Leu Phe Glu Gln Tyr Ile Asn
 210 215 220
 Asn Leu Arg Arg Gln Leu Asp Ser Ile Val Gly Glu Arg Gly Arg Leu
 225 230 235 240
 Asp Ser Glu Leu Arg Asn Met Gln Asp Leu Val Glu Asp Phe Lys Asn
 245 250 255
 Lys Tyr Glu Asp Glu Ile Asn Lys Arg Thr Thr Ala Glu Asn Glu Phe
 260 265 270
 Val Met Leu Lys Lys Asp Val Asp Ala Ala Tyr Met Asn Lys Val Glu
 275 280 285

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Leu Glu Ala Lys Val Asp Ala Leu Met Asp Glu Ile Asn Phe Met Lys
 290 295 300
 Met Phe Phe Asp Ala Glu Leu Ser Gln Met Gln Thr His Val Ser Asp
 305 310 315 320
 Thr Ser Val Val Leu Ser Met Asp Asn Asn Arg Asn Leu Asp Leu Asp
 325 330 335
 Ser Ile Ile Ala Glu Val Lys Ala Gln Tyr Glu Glu Ile Ala Asn Arg
 340 345 350
 Ser Arg Thr Glu Ala Glu Ser Trp Tyr Gln Thr Lys Tyr Glu Glu Leu
 355 360 365
 Gln Gln Thr Ala Gly Arg His Gly Asp Asp Leu Arg Asn Thr Lys His
 370 375 380
 Glu Ile Thr Glu Met Asn Arg Met Ile Gln Arg Leu Arg Ala Glu Ile
 385 390 395 400
 Asp Asn Val Lys Lys Gln Cys Ala Asn Leu Gln Asn Ala Ile Ala Asp
 405 410 415
 Ala Glu Gln Arg Gly Glu Leu Ala Leu Lys Asp Ala Arg Asn Lys Leu
 420 425 430
 Ala Glu Leu Glu Glu Ala Leu Gln Lys Ala Lys Gln Asp Met Ala Arg
 435 440 445
 Leu Leu Arg Glu Tyr Gln Glu Leu Met Asn Thr Lys Leu Ala Leu Asp
 450 455 460
 Val Glu Ile Ala Thr Tyr Arg Lys Leu Leu Glu Gly Glu Glu Cys Arg
 465 470 475 480
 Leu Ser Gly Glu Gly Val Gly Pro Val Asn Ile Ser Val Val Thr Ser
 485 490 495
 Ser Val Ser Ser Gly Tyr Gly Ser Gly Ser Gly Tyr Gly Gly Leu
 500 505 510
 Gly Gly Gly Leu Gly Gly Gly Leu Gly Gly Gly Leu Ala Gly Gly Ser
 515 520 525
 Ser Gly Ser Tyr Tyr Ser Ser Ser Gly Gly Val Gly Leu Gly Gly
 530 535 540
 Gly Leu Ser Val Gly Gly Ser Gly Phe Ser Ala Ser Ser Gly Arg Gly
 545 550 555 560
 Leu Gly Val Gly Phe Gly Ser Gly Gly Gly Ser Ser Ser Ser Val Lys
 565 570 575
 Phe Val Ser Thr Ser Ser Ser Arg Lys Ser Phe Lys Ser
 580 585 590

<210> 458

<211> 523

<212> PRT

<213> Homo sapiens

<400> 458

Met Ser Arg Gln Phe Thr Cys Lys Ser Gly Ala Ala Ala Lys Gly Gly
 1 5 10 15
 Phe Ser Gly Cys Ser Ala Val Leu Ser Gly Gly Ser Ser Ser Ser Phe
 20 25 30
 Arg Ala Gly Ser Lys Gly Leu Ser Gly Gly Phe Gly Ser Arg Ser Leu
 35 40 45
 Tyr Ser Leu Gly Gly Val Arg Ser Leu Asn Val Ala Ser Gly Ser Gly
 50 55 60
 Lys Ser Gly Gly Tyr Gly Phe Gly Arg Gly Arg Ala Ser Gly Phe Ala
 65 70 75 80
 Gly Ser Met Phe Gly Ser Val Ala Leu Gly Pro Val Cys Pro Thr Val
 85 90 95
 Cys Pro Pro Gly Gly Ile His Gln Val Thr Val Asn Glu Ser Leu Leu
 100 105 110
 Ala Pro Leu Asn Val Glu Leu Asp Pro Glu Ile Gln Lys Val Arg Ala
 115 120 125

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Gln Glu Arg Glu Gln Ile Lys Ala Leu Asn Asn Lys Phe Ala Ser Phe
 130 135 140
 Ile Asp Lys Val Arg Phe Leu Glu Gln Gln Asn Gln Val Leu Glu Thr
 145 150 155 160
 Lys Trp Glu Leu Leu Gln Gln Leu Asp Leu Asn Asn Cys Lys Asn Asn
 165 170 175
 Leu Glu Pro Ile Leu Glu Gly Tyr Ile Ser Asn Leu Arg Lys Gln Leu
 180 185 190
 Glu Thr Leu Ser Gly Asp Arg Val Arg Leu Asp Ser Glu Leu Arg Asn
 195 200 205
 Val Arg Asp Val Val Glu Asp Tyr Lys Lys Arg Tyr Glu Glu Glu Ile
 210 215 220
 Asn Lys Arg Thr Ala Ala Glu Asn Glu Phe Val Leu Leu Lys Lys Asp
 225 230 235 240
 Val Asp Ala Ala Tyr Ala Asn Lys Val Glu Leu Gln Ala Lys Val Glu
 245 250 255
 Ser Met Asp Gln Glu Ile Lys Phe Phe Arg Cys Leu Phe Glu Ala Glu
 260 265 270
 Ile Thr Gln Ile Gln Ser His Ile Ser Asp Met Ser Val Ile Leu Ser
 275 280 285
 Met Asp Asn Asn Arg Asn Leu Asp Leu Asp Ser Ile Ile Asp Glu Val
 290 295 300
 Arg Thr Gln Tyr Glu Glu Ile Ala Leu Lys Ser Lys Ala Glu Ala Glu
 305 310 315 320
 Ala Leu Tyr Gln Thr Lys Phe Gln Glu Leu Gln Leu Ala Ala Gly Arg
 325 330 335
 His Gly Asp Asp Leu Lys Asn Thr Lys Asn Glu Ile Ser Glu Leu Thr
 340 345 350
 Arg Leu Ile Gln Arg Ile Arg Ser Glu Ile Glu Asn Val Lys Lys Gln
 355 360 365
 Ala Ser Asn Leu Glu Thr Ala Ile Ala Asp Ala Glu Gln Arg Gly Asp
 370 375 380
 Asn Ala Leu Lys Asp Ala Arg Ala Lys Leu Asp Glu Leu Glu Gly Ala
 385 390 395 400
 Leu His Gln Ala Lys Glu Glu Leu Ala Arg Met Leu Arg Glu Tyr Gln
 405 410 415
 Glu Leu Met Ser Leu Lys Leu Ala Leu Asp Met Glu Ile Ala Thr Tyr
 420 425 430
 Arg Lys Leu Leu Glu Ser Glu Glu Cys Arg Met Ser Gly Glu Phe Pro
 435 440 445
 Ser Pro Val Ser Ile Ser Ile Ile Ser Ser Thr Ser Gly Gly Ser Val
 450 455 460
 Tyr Gly Phe Arg Pro Ser Met Val Ser Gly Tyr Val Ala Asn Ser
 465 470 475 480
 Ser Asn Cys Ile Ser Gly Val Cys Ser Val Arg Gly Gly Glu Gly Arg
 485 490 495
 Ser Arg Gly Ser Ala Asn Asp Tyr Lys Asp Thr Leu Gly Lys Gly Ser
 500 505 510
 Ser Leu Ser Ala Pro Ser Lys Lys Thr Ser Arg
 515 520
 <210> 459
 <211> 529
 <212> PRT
 <213> Homo sapiens
 <400> 459
 Met Ser Arg Gln Leu Asn Ile Lys Ser Ser Gly Asp Lys Gly Asn Phe
 1 5 10 15
 Ser Val His Ser Ala Val Val Pro Arg Lys Ala Val Gly Ser Leu Ala
 20 25 30

Ser Tyr Cys Ala Ala Gly Arg Gly Ala Gly Ala Gly Phe Gly Ser Arg
 35 40 45
 Ser Leu Tyr Ser Leu Gly Gly Asn Arg Arg Ile Ser Phe Asn Val Ala
 50 55 60
 Gly Gly Gly Val Arg Ala Gly Gly Tyr Gly Phe Arg Pro Gly Ser Gly
 65 70 75 80
 Tyr Gly Gly Gly Arg Ala Ser Gly Phe Ala Gly Ser Met Phe Gly Ser
 85 90 95
 Val Ala Leu Gly Pro Ala Cys Leu Ser Val Cys Pro Pro Gly Ile
 100 105 110
 His Gln Val Thr Val Asn Lys Ser Leu Leu Ala Pro Leu Asn Val Glu
 115 120 125
 Leu Asp Pro Glu Ile Gln Lys Val Arg Ala Gln Glu Arg Glu Gln Ile
 130 135 140
 Lys Val Leu Asn Asp Lys Phe Ala Ser Phe Ile Asp Lys Val Arg Phe
 145 150 155 160
 Leu Glu Gln Gln Asn Gln Val Leu Glu Thr Lys Trp Glu Leu Leu Gln
 165 170 175
 Gln Leu Asp Leu Asn Asn Cys Lys Lys Asn Leu Glu Pro Ile Leu Glu
 180 185 190
 Gly Tyr Ile Ser Asn Leu Arg Lys Gln Leu Glu Thr Leu Ser Gly Asp
 195 200 205
 Arg Val Arg Leu Asp Ser Glu Leu Arg Ser Met Arg Asp Leu Val Glu
 210 215 220
 Asp Tyr Lys Lys Arg Tyr Glu Val Glu Ile Asn Arg Arg Thr Thr Ala
 225 230 235 240
 Glu Asn Glu Phe Val Val Leu Lys Lys Asp Ala Asp Ala Tyr Ala
 245 250 255
 Val Lys Val Glu Leu Gln Ala Lys Val Asp Ser Leu Asp Lys Asp Ile
 260 265 270
 Lys Phe Leu Lys Cys Leu Tyr Asp Ala Glu Ile Ala Gln Ile Gln Thr
 275 280 285
 His Ala Ser Glu Thr Ser Val Ile Leu Ser Met Asp Asn Asn Arg Asp
 290 295 300
 Leu Asp Leu Asp Ser Ile Ile Ala Glu Val Arg Met His Tyr Glu Glu
 305 310 315 320
 Ile Ala Leu Lys Ser Lys Ala Glu Ala Glu Ala Leu Tyr Gln Thr Lys
 325 330 335
 Ile Gln Glu Leu Gln Leu Ala Ala Ser Arg His Gly Asp Asp Leu Lys
 340 345 350
 His Thr Arg Ser Glu Met Val Glu Leu Asn Arg Leu Ile Gln Arg Ile
 355 360 365
 Arg Cys Glu Ile Gly Asn Val Lys Lys Gln Arg Ala Ser Leu Glu Thr
 370 375 380
 Ala Ile Ala Asp Ala Glu Gln Arg Gly Asp Asn Ala Leu Lys Asp Ala
 385 390 395 400
 Gln Ala Lys Leu Asp Glu Leu Glu Gly Ala Leu His Gln Ala Lys Glu
 405 410 415
 Glu Leu Ala Arg Met Leu Arg Glu Tyr Gln Glu Leu Met Ser Leu Lys
 420 425 430
 Leu Ala Leu Asp Met Glu Ile Ala Thr Tyr Arg Lys Leu Leu Glu Gly
 435 440 445
 Glu Glu Cys Arg Met Ser Gly Glu Asn Pro Ser Ser Val Ser Ile Ser
 450 455 460
 Val Ile Ser Ser Ser Ser Tyr Ser Tyr His His Pro Ser Ser Ala Gly
 465 470 475 480
 Val Asp Leu Gly Ala Ser Ala Val Ala Gly Ser Ser Gly Ser Thr Gln
 485 490 495
 Ser Gly Gln Thr Lys Thr Thr Glu Ala Arg Gly Gly Asp Leu Lys Asp
 500 505 510
 Thr Gln Gly Lys Ser Thr Pro Ala Ser Ile Pro Ala Arg Lys Ala Thr
 515 520 525
 Arg

<211> 511

<212> PRT

<213> Homo sapiens

<400> 460

Met	Ser	Arg	Gln	Leu	Thr	His	Phe	Pro	Arg	Gly	Glu	Arg	Leu	Gly	Phe
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Ser	Gly	Cys	Ser	Ala	Val	Leu	Ser	Gly	Gly	Ile	Gly	Ser	Ser	Ser	Ala
			20					25					30		
Ser	Phe	Arg	Ala	Arg	Val	Lys	Gly	Ser	Ala	Ser	Phe	Gly	Ser	Lys	Ser
			35				40					45			
Leu	Ser	Cys	Leu	Gly	Gly	Ser	Arg	Ser	Leu	Ala	Leu	Ser	Ala	Ala	Ala
			50			55					60				
Arg	Arg	Gly	Gly	Gly	Arg	Leu	Gly	Gly	Phe	Val	Gly	Thr	Ala	Phe	Gly
65					70				75					80	
Ser	Ala	Gly	Leu	Gly	Pro	Lys	Cys	Pro	Ser	Val	Cys	Pro	Pro	Gly	Gly
				85					90					95	
Ile	Pro	Gln	Val	Thr	Val	Asn	Lys	Ser	Leu	Leu	Ala	Pro	Leu	Asn	Val
			100					105					110		
Glu	Met	Asp	Pro	Glu	Ile	Gln	Arg	Val	Arg	Ala	Gln	Glu	Arg	Glu	Gln
			115				120					125			
Ile	Lys	Ala	Leu	Asn	Asn	Lys	Phe	Ala	Ser	Phe	Ile	Asp	Lys	Val	Arg
			130				135					140			
Phe	Leu	Glu	Gln	Gln	Asn	Gln	Val	Leu	Glu	Thr	Lys	Trp	Asn	Leu	Leu
145					150					155				160	
Gln	Gln	Leu	Asp	Leu	Asn	Asn	Cys	Arg	Lys	Asn	Leu	Glu	Pro	Ile	Tyr
				165					170					175	
Glu	Gly	Tyr	Ile	Ser	Asn	Leu	Gln	Lys	Gln	Leu	Glu	Met	Leu	Ser	Gly
			180					185					190		
Asp	Gly	Val	Arg	Leu	Asp	Ser	Glu	Leu	Arg	Asn	Met	Gln	Asp	Leu	Val
			195				200					205			
Glu	Asp	Tyr	Lys	Lys	Arg	Tyr	Glu	Val	Glu	Ile	Asn	Arg	Arg	Thr	Ala
			210			215					220				
Ala	Glu	Asn	Glu	Phe	Val	Val	Leu	Lys	Lys	Asp	Val	Asp	Ala	Ala	Tyr
225					230					235				240	
Met	Asn	Lys	Val	Glu	Leu	Gln	Ala	Lys	Val	Asp	Ser	Leu	Thr	Asp	Glu
				245					250					255	
Ile	Lys	Phe	Phe	Lys	Cys	Leu	Tyr	Glu	Gly	Glu	Ile	Thr	Gln	Ile	Gln
			260					265					270		
Ser	His	Ile	Ser	Asp	Thr	Ser	Ile	Val	Leu	Ser	Met	Asp	Asn	Asn	Arg
			275				280					285			
Asp	Leu	Asp	Leu	Asp	Ser	Ile	Ile	Ala	Glu	Val	Arg	Ala	Gln	Tyr	Glu
			290			295					300				
Glu	Ile	Ala	Leu	Lys	Ser	Lys	Ala	Glu	Ala	Glu	Thr	Leu	Tyr	Gln	Thr
305					310					315				320	
Lys	Ile	Gln	Glu	Leu	Gln	Val	Thr	Ala	Gly	Gln	His	Gly	Asp	Asp	Leu
				325					330					335	
Lys	Leu	Thr	Lys	Ala	Glu	Ile	Ser	Glu	Leu	Asn	Arg	Leu	Ile	Gln	Arg
			340					345					350		
Ile	Arg	Ser	Glu	Ile	Gly	Asn	Val	Lys	Lys	Gln	Cys	Ala	Asp	Leu	Glu
			355			360						365			
Thr	Ala	Ile	Ala	Asp	Ala	Glu	Gln	Arg	Gly	Asp	Cys	Ala	Leu	Lys	Asp
			370			375					380				
Ala	Arg	Ala	Lys	Leu	Asp	Glu	Leu	Glu	Gly	Ala	Leu	His	Gln	Ala	Lys
385					390					395				400	
Glu	Glu	Leu	Ala	Arg	Met	Leu	Arg	Glu	Tyr	Gln	Glu	Leu	Val	Ser	Leu
				405					410					415	
Lys	Leu	Ala	Leu	Asp	Met	Glu	Ile	Ala	Thr	Tyr	Arg	Lys	Leu	Leu	Glu
			420					425					430		
Ser	Glu	Glu	Cys	Arg	Met	Ser	Gly	Glu	Tyr	Pro	Asn	Ser	Val	Ser	Ile
			435				440					445			

Ser Val Ile Ser Ser Thr Asn Ala Gly Ala Gly Gly Ala Gly Phe Ser
 450 455 460
 Met Gly Phe Gly Ala Ser Ser Tyr Ser Tyr Lys Thr Ala Ala Ala
 465 470 475 480
 Asp Val Lys Thr Lys Gly Ser Cys Gly Ser Glu Leu Lys Asp Pro Leu
 485 490 495
 Ala Lys Thr Ser Gly Ser Ser Cys Ala Thr Lys Lys Ala Ser Arg
 500 505 510
 <210> 461
 <211> 540
 <212> PRT
 <213> Homo sapiens

<400> 461
 Met Ser Arg Gln Phe Thr Tyr Lys Ser Gly Ala Ala Ala Lys Gly Gly
 1 5 10 15
 Phe Ser Gly Cys Ser Ala Val Leu Ser Gly Gly Ser Ser Ser Tyr
 20 25 30
 Arg Ala Gly Gly Lys Gly Leu Ser Gly Gly Phe Ser Ser Arg Ser Leu
 35 40 45
 Tyr Ser Leu Gly Gly Ala Arg Ser Ile Ser Phe Asn Val Ala Ser Gly
 50 55 60
 Ser Gly Trp Ala Gly Gly Tyr Gly Phe Gly Arg Gly Arg Ala Ser Gly
 65 70 75 80
 Phe Ala Gly Ser Met Phe Gly Ser Val Ala Leu Gly Ser Val Cys Pro
 85 90 95
 Ser Leu Cys Pro Pro Gly Gly Ile His Gln Val Thr Ile Asn Lys Ser
 100 105 110
 Leu Leu Ala Pro Leu Asn Val Glu Leu Asp Pro Glu Ile Gln Lys Val
 115 120 125
 Arg Ala Gln Glu Arg Glu Gln Ile Lys Val Leu Asn Asn Lys Phe Ala
 130 135 140
 Ser Phe Ile Asp Lys Val Arg Phe Leu Glu Gln Asn Gln Val Leu
 145 150 155 160
 Glu Thr Lys Trp Glu Leu Leu Gln Gln Leu Asp Leu Asn Asn Cys Lys
 165 170 175
 Asn Asn Leu Glu Pro Ile Leu Glu Gly Tyr Ile Ser Asn Leu Arg Lys
 180 185 190
 Gln Leu Glu Thr Leu Ser Gly Asp Arg Val Arg Leu Asp Ser Glu Leu
 195 200 205
 Arg Ser Val Arg Glu Val Val Glu Asp Tyr Lys Lys Arg Tyr Glu Glu
 210 215 220
 Glu Ile Asn Lys Arg Thr Thr Ala Glu Asn Glu Phe Val Val Leu Lys
 225 230 235 240
 Lys Asp Val Asp Ala Ala Tyr Thr Ser Lys Val Glu Leu Gln Ala Lys
 245 250 255
 Val Asp Ala Leu Asp Gly Glu Ile Lys Phe Phe Lys Cys Leu Tyr Glu
 260 265 270
 Gly Glu Thr Ala Gln Ile Gln Ser His Ile Ser Asp Thr Ser Ile Ile
 275 280 285
 Leu Ser Met Asp Asn Asn Arg Asn Leu Asp Leu Asp Ser Ile Ile Ala
 290 295 300
 Glu Val Arg Ala Gln Tyr Glu Glu Ile Ala Arg Lys Ser Lys Ala Glu
 305 310 315 320
 Ala Glu Ala Leu Tyr Gln Thr Lys Phe Gln Glu Leu Gln Leu Ala Ala
 325 330 335
 Gly Arg His Gly Asp Asp Leu Lys His Thr Lys Asn Glu Ile Ser Glu
 340 345 350
 Leu Thr Arg Leu Ile Gln Arg Leu Arg Ser Glu Ile Glu Ser Val Lys
 355 360 365

Lys Gln Cys Ala Asn Leu Glu Thr Ala Ile Ala Asp Ala Glu Gln Arg
 370 375 380
 Gly Asp Cys Ala Leu Lys Asp Ala Arg Ala Lys Leu Asp Glu Leu Glu
 385 390 395 400
 Gly Ala Leu Gln Gln Ala Lys Glu Glu Leu Ala Arg Met Leu Arg Glu
 405 410 415
 Tyr Gln Glu Leu Leu Ser Val Lys Leu Ser Leu Asp Ile Glu Ile Ala
 420 425 430
 Thr Tyr Arg Lys Leu Leu Glu Gly Glu Glu Cys Arg Met Ser Gly Glu
 435 440 445
 Tyr Thr Asn Ser Val Ser Ile Ser Val Ile Asn Ser Ser Met Ala Gly
 450 455 460
 Met Ala Gly Thr Gly Ala Gly Phe Gly Phe Ser Asn Ala Gly Thr Tyr
 465 470 475 480
 Gly Tyr Trp Pro Ser Ser Val Ser Gly Gly Tyr Ser Met Leu Pro Gly
 485 490 495
 Gly Cys Val Thr Gly Ser Gly Asn Cys Ser Pro Arg Gly Glu Ala Arg
 500 505 510
 Thr Arg Leu Gly Ser Ala Ser Glu Phe Arg Asp Ser Gln Gly Lys Thr
 515 520 525
 Leu Ala Leu Ser Ser Pro Thr Lys Lys Thr Met Arg
 530 535 540
 <210> 462
 <211> 645
 <212> PRT
 <213> Homo sapiens

<400> 462
 Met Ser Cys Gln Ile Ser Cys Lys Ser Arg Gly Arg Gly Gly Gly Gly
 1 5 10 15
 Gly Gly Phe Arg Gly Phe Ser Ser Gly Ser Ala Val Val Ser Gly Gly
 20 25 30
 Ser Arg Arg Ser Thr Ser Ser Phe Ser Cys Leu Ser Arg His Gly Gly
 35 40 45
 Gly Gly Gly Gly Phe Gly Gly Gly Gly Phe Gly Ser Arg Ser Leu Val
 50 55 60
 Gly Leu Gly Gly Thr Lys Ser Ile Ser Ile Ser Val Ala Gly Gly Gly
 65 70 75 80
 Gly Gly Phe Gly Ala Gly Gly Phe Gly Gly Arg Gly Gly Gly Phe
 85 90 95
 Gly Gly Gly Ser Gly Phe Gly Gly Gly Ser Gly Phe Gly Gly Gly Ser
 100 105 110
 Gly Phe Ser Gly Gly Gly Phe Gly Gly Gly Phe Gly Gly Gly Arg
 115 120 125
 Phe Gly Gly Phe Gly Gly Pro Gly Gly Val Gly Gly Leu Gly Gly Pro
 130 135 140
 Gly Gly Phe Gly Pro Gly Gly Tyr Pro Gly Gly Ile His Glu Val Ser
 145 150 155 160
 Val Asn Gln Ser Leu Leu Gln Pro Leu Asn Val Lys Val Asp Pro Glu
 165 170 175
 Ile Gln Asn Val Lys Ala Gln Glu Arg Glu Gln Ile Lys Thr Leu Asn
 180 185 190
 Asn Lys Phe Ala Ser Phe Ile Asp Lys Val Arg Phe Leu Glu Gln Gln
 195 200 205
 Asn Gln Val Leu Gln Thr Lys Trp Glu Leu Leu Gln Gln Met Asn Val
 210 215 220
 Gly Thr Arg Pro Ile Asn Leu Glu Pro Ile Phe Gln Gly Tyr Ile Asp
 225 230 235 240
 Ser Leu Lys Arg Tyr Leu Asp Gly Leu Thr Ala Glu Arg Thr Ser Gln
 245 250 255

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Asn	Ser	Glu	Leu	Asn	Asn	Met	Gln	Asp	Leu	Val	Glu	Asp	Tyr	Lys	Lys	
			260					265					270			
Lys	Tyr	Glu	Asp	Glu	Ile	Asn	Lys	Arg	Thr	Ala	Ala	Glu	Asn	Asp	Phe	
		275					280					285				
Val	Thr	Leu	Lys	Lys	Asp	Val	Asp	Asn	Ala	Tyr	Met	Ile	Lys	Val	Glu	
	290					295					300					
Leu	Gln	Ser	Lys	Val	Asp	Leu	Leu	Asn	Gln	Glu	Ile	Glu	Phe	Leu	Lys	
305					310					315					320	
Val	Leu	Tyr	Asp	Ala	Glu	Ile	Ser	Gln	Ile	His	Gln	Ser	Val	Thr	Asp	
				325					330						335	
Thr	Asn	Val	Ile	Leu	Ser	Met	Asp	Asn	Ser	Arg	Asn	Leu	Asp	Leu	Asp	
			340					345					350			
Ser	Ile	Ile	Ala	Glu	Val	Lys	Ala	Gln	Tyr	Glu	Glu	Ile	Ala	Gln	Arg	
		355					360					365				
Ser	Lys	Glu	Glu	Ala	Glu	Ala	Leu	Tyr	His	Ser	Lys	Tyr	Glu	Glu	Leu	
	370					375					380					
Gln	Val	Thr	Val	Gly	Arg	His	Gly	Asp	Ser	Leu	Lys	Glu	Ile	Lys	Ile	
385					390					395					400	
Glu	Ile	Ser	Glu	Leu	Asn	Arg	Val	Ile	Gln	Arg	Leu	Gln	Gly	Glu	Ile	
				405					410						415	
Ala	His	Val	Lys	Gln	Cys	Lys	Asn	Val	Gln	Asp	Ala	Ile	Ala	Asp		
		420					425					430				
Ala	Glu	Gln	Arg	Gly	Glu	His	Ala	Leu	Lys	Asp	Ala	Arg	Asn	Lys	Leu	
		435					440					445				
Asn	Asp	Leu	Glu	Glu	Ala	Leu	Gln	Gln	Ala	Lys	Glu	Asp	Leu	Ala	Arg	
	450					455					460					
Leu	Leu	Arg	Asp	Tyr	Gln	Glu	Leu	Met	Asn	Val	Lys	Leu	Ala	Leu	Asp	
465					470					475					480	
Val	Glu	Ile	Ala	Thr	Tyr	Arg	Lys	Leu	Leu	Glu	Gly	Glu	Glu	Cys	Arg	
			485						490					495		
Met	Ser	Gly	Asp	Leu	Ser	Ser	Asn	Val	Thr	Val	Ser	Val	Thr	Ser	Ser	
			500					505					510			
Thr	Ile	Ser	Ser	Asn	Val	Ala	Ser	Lys	Ala	Ala	Phe	Gly	Gly	Ser	Gly	
		515					520					525				
Gly	Arg	Gly	Ser	Ser	Ser	Gly	Gly	Gly	Tyr	Ser	Ser	Gly	Ser	Ser	Ser	
	530					535					540					
Tyr	Gly	Ser	Gly	Gly	Arg	Gln	Ser	Gly	Ser	Arg	Gly	Gly	Ser	Gly	Gly	
545					550					555					560	
Gly	Gly	Ser	Ile	Ser	Gly	Gly	Gly	Tyr	Gly	Ser	Gly	Gly	Gly	Ser	Gly	
			565						570					575		
Gly	Arg	Tyr	Gly	Ser	Gly	Gly	Gly	Ser	Lys	Gly	Gly	Ser	Ile	Ser	Gly	
			580					585					590			
Gly	Gly	Tyr	Gly	Ser	Gly	Gly	Gly	Lys	His	Ser	Ser	Gly	Gly	Gly	Ser	
		595					600					605				
Arg	Gly	Gly	Ser	Ser	Ser	Gly	Gly	Gly	Tyr	Gly	Ser	Gly	Gly	Gly	Gly	
	610					615					620					
Ser	Ser	Ser	Val	Lys	Gly	Ser	Ser	Gly	Glu	Ala	Phe	Gly	Ser	Ser	Val	
625					630					635					640	
Thr	Phe	Ser	Phe	Arg												
				645												

<210> 463

<211> 644

<212> PRT

<213> Homo sapiens

<400> 463

Met	Ser	Arg	Gln	Phe	Ser	Ser	Arg	Ser	Gly	Tyr	Arg	Ser	Gly	Gly	Gly	
1			5						10				15			
Phe	Ser	Ser	Gly	Ser	Ala	Gly	Ile	Ile	Asn	Tyr	Gln	Arg	Arg	Thr	Thr	
			20					25					30			

Ser	Ser	Ser	Thr	Arg	Arg	Ser	Gly	Gly	Gly	Gly	Gly	Arg	Phe	Ser	Ser
		35					40					45			
Cys	Gly	Gly	Gly	Gly	Gly	Ser	Phe	Gly	Ala	Gly	Gly	Gly	Phe	Gly	Ser
	50					55					60				
Arg	Ser	Leu	Val	Asn	Leu	Gly	Gly	Ser	Lys	Ser	Ile	Ser	Ile	Ser	Val
65					70					75					80
Ala	Arg	Gly	Gly	Gly	Arg	Gly	Ser	Gly	Phe	Gly	Gly	Gly	Tyr	Gly	Gly
				85					90				95		
Gly	Gly	Phe	Gly	Gly	Gly	Gly	Phe	Gly	Gly	Gly	Gly	Phe	Gly	Gly	Gly
			100					105					110		
Gly	Ile	Gly	Gly	Gly	Gly	Phe	Gly	Gly	Phe	Gly	Ser	Gly	Gly	Gly	Gly
		115					120					125			
Phe	Gly	Gly	Gly	Gly	Phe	Gly	Gly	Gly	Gly	Tyr	Gly	Gly	Gly	Tyr	Gly
	130					135					140				
Pro	Val	Cys	Pro	Pro	Gly	Gly	Ile	Gln	Glu	Val	Thr	Ile	Asn	Gln	Ser
145					150					155					160
Leu	Leu	Gln	Pro	Leu	Asn	Val	Glu	Ile	Asp	Pro	Glu	Ile	Gln	Lys	Val
				165					170					175	
Lys	Ser	Arg	Glu	Arg	Glu	Gln	Ile	Lys	Ser	Leu	Asn	Asn	Gln	Phe	Ala
			180					185					190		
Ser	Phe	Ile	Asp	Lys	Val	Arg	Phe	Leu	Glu	Gln	Gln	Asn	Gln	Val	Leu
		195					200						205		
Gln	Thr	Lys	Trp	Glu	Leu	Leu	Gln	Gln	Val	Asp	Thr	Ser	Thr	Arg	Thr
	210					215					220				
His	Asn	Leu	Glu	Pro	Tyr	Phe	Glu	Ser	Phe	Ile	Asn	Asn	Leu	Arg	Arg
225					230					235					240
Arg	Val	Asp	Gln	Leu	Lys	Ser	Asp	Gln	Ser	Arg	Leu	Asp	Ser	Glu	Leu
			245						250					255	
Lys	Asn	Met	Gln	Asp	Met	Val	Glu	Asp	Tyr	Arg	Asn	Lys	Tyr	Glu	Asp
		260						265					270		
Glu	Ile	Asn	Lys	Arg	Thr	Asn	Ala	Glu	Asn	Glu	Phe	Val	Thr	Ile	Lys
		275					280					285			
Lys	Asp	Val	Asp	Gly	Ala	Tyr	Met	Thr	Lys	Val	Asp	Leu	Gln	Ala	Lys
	290					295					300				
Leu	Asp	Asn	Leu	Gln	Gln	Glu	Ile	Asp	Phe	Leu	Thr	Ala	Leu	Tyr	Gln
305					310					315					320
Ala	Glu	Leu	Ser	Gln	Met	Gln	Thr	Gln	Ile	Ser	Glu	Thr	Asn	Val	Ile
				325					330					335	
Leu	Ser	Met	Asp	Asn	Asn	Arg	Ser	Leu	Asp	Leu	Asp	Ser	Ile	Ile	Ala
		340						345					350		
Glu	Val	Lys	Ala	Gln	Tyr	Glu	Asp	Ile	Ala	Gln	Lys	Ser	Lys	Ala	Glu
		355					360					365			
Ala	Glu	Ser	Leu	Tyr	Gln	Ser	Lys	Tyr	Glu	Glu	Leu	Gln	Ile	Thr	Ala
	370					375					380				
Gly	Arg	His	Gly	Asp	Ser	Val	Arg	Asn	Ser	Lys	Ile	Glu	Ile	Ser	Glu
385					390					395					400
Leu	Asn	Arg	Val	Ile	Gln	Arg	Leu	Arg	Ser	Glu	Ile	Asp	Asn	Val	Lys
				405					410					415	
Lys	Gln	Ile	Ser	Asn	Leu	Gln	Gln	Ser	Ile	Ser	Asp	Ala	Glu	Gln	Arg
		420						425					430		
Gly	Glu	Asn	Ala	Leu	Lys	Asp	Ala	Lys	Asn	Lys	Leu	Asn	Asp	Leu	Glu
		435					440					445			
Asp	Ala	Leu	Gln	Gln	Ala	Lys	Glu	Asp	Leu	Ala	Arg	Leu	Leu	Arg	Asp
	450					455					460				
Tyr	Gln	Glu	Leu	Met	Asn	Thr	Lys	Leu	Ala	Leu	Asp	Leu	Glu	Ile	Ala
465					470					475					480
Thr	Tyr	Arg	Thr	Leu	Leu	Glu	Gly	Glu	Glu	Ser	Arg	Met	Ser	Gly	Glu
				485					490					495	
Cys	Ala	Pro	Asn	Val	Ser	Val	Ser	Val	Ser	Thr	Ser	His	Thr	Thr	Ile
			500					505					510		
Ser	Gly	Gly	Gly	Ser	Arg	Gly	Gly	Gly	Gly	Gly	Gly	Tyr	Gly	Ser	Gly
		515					520					525			
Gly	Ser	Ser	Tyr	Gly	Ser	Gly	Gly	Gly	Ser	Tyr	Gly	Ser	Gly	Gly	Gly
	530					535					540				
Gly	Gly	Gly	Gly	Arg	Gly	Ser	Tyr	Gly	Ser	Gly	Gly	Ser	Ser	Tyr	Gly
545					550					555					560

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[illegible]

<210> 464

<211> 629

<212> PRT

<213> Homo sapiens

<400>	464														
Met	Ser	Arg	Gln	Ala	Ser	Lys	Thr	Ser	Gly	Gly	Gly	Ser	Gln	Gly	Phe
1			5						10					15	
Ser	Gly	Arg	Ser	Ala	Val	Val	Ser	Gly	Ser	Ser	Arg	Met	Ser	Cys	Val
			20					25					30		
Ala	His	Ser	Gly	Gly	Ala	Gly	Gly	Gly	Ala	Tyr	Gly	Phe	Arg	Ser	Gly
		35					40					45			
Ala	Gly	Gly	Phe	Gly	Ser	Arg	Ser	Leu	Tyr	Asn	Leu	Gly	Gly	Asn	Lys
	50				55					60					
Ser	Ile	Ser	Ile	Ser	Val	Ala	Ala	Gly	Gly	Ser	Arg	Ala	Gly	Gly	Phe
65					70					75					80
Gly	Gly	Gly	Arg	Ser	Ser	Cys	Ala	Phe	Ala	Gly	Gly	Tyr	Gly	Gly	Gly
			85						90					95	
Phe	Gly	Ser	Gly	Tyr	Gly	Gly	Gly	Phe	Gly	Gly	Gly	Phe	Gly	Gly	Gly
			100					105					110		
Arg	Gly	Met	Gly	Gly	Gly	Phe	Gly	Gly	Ala	Gly	Gly	Phe	Gly	Gly	Ala
		115					120					125			
Gly	Gly	Phe	Gly	Gly	Ala	Gly	Gly	Phe	Gly	Gly	Pro	Gly	Gly	Phe	Gly
			130			135					140				
Gly	Ser	Gly	Gly	Phe	Gly	Gly	Pro	Gly	Ser	Leu	Gly	Ser	Pro	Gly	Gly
145					150					155					160
Phe	Ala	Pro	Gly	Gly	Phe	Pro	Gly	Gly	Ile	Gln	Glu	Val	Thr	Thr	Asn
			165						170					175	
Gln	Ser	Leu	Leu	Gln	Pro	Leu	Lys	Val	Glu	Thr	Asp	Pro	Gln	Ile	Gly
			180					185					190		
Gln	Val	Lys	Ala	Gln	Glu	Arg	Glu	Gln	Ile	Lys	Thr	Leu	Asn	Asn	Lys
		195					200					205			
Phe	Ala	Ser	Phe	Ile	Asp	Lys	Val	Arg	Phe	Leu	Glu	Gln	Gln	Asn	Lys
	210				215						220				
Val	Leu	Glu	Thr	Lys	Trp	Asn	Leu	Leu	Gln	Gln	Gln	Gly	Thr	Ser	Ser
225					230					235					240
Ile	Ser	Gly	Thr	Asn	Asn	Leu	Glu	Pro	Leu	Phe	Glu	Asn	His	Ile	Asn
			245						250					255	
Tyr	Leu	Arg	Ser	Tyr	Leu	Asp	Asn	Ile	Leu	Gly	Glu	Arg	Gly	Arg	Leu
			260					265					270		
Asp	Ser	Glu	Leu	Lys	Asn	Met	Glu	Asp	Leu	Val	Glu	Asp	Phe	Lys	Lys
		275					280					285			
Lys	Tyr	Glu	Asp	Glu	Ile	Asn	Lys	Arg	Thr	Ala	Ala	Glu	Asn	Glu	Phe
			290			295					300				
Val	Thr	Leu	Lys	Lys	Asp	Val	Asp	Ser	Ala	Tyr	Met	Asn	Lys	Val	Glu
305					310					315					320
Leu	Gln	Ala	Lys	Val	Asp	Ala	Leu	Ile	Asp	Glu	Ile	Asp	Phe	Leu	Arg
			325						330					335	

Thr Leu Tyr Asp Ala Glu Leu Ser Gln Met Gln Ser His Ile Ser Asp
 340 345 350
 Thr Ser Val Val Leu Ser Met Asp Asn Asn Arg Ser Leu Asp Leu Asp
 355 360 365
 Ser Ile Ile Ala Glu Val Gly Ala Gln Tyr Glu Asp Ile Ala Gln Arg
 370 375 380
 Ser Lys Ala Glu Ala Glu Ala Leu Tyr Gln Thr Lys Leu Gly Glu Leu
 385 390 395 400
 Gln Thr Thr Ala Gly Arg His Gly Asp Asp Leu Arg Asn Thr Lys Ser
 405 410 415
 Glu Ile Ile Glu Leu Asn Arg Met Ile Gln Arg Leu Arg Ala Glu Ile
 420 425 430
 Glu Gly Val Lys Lys Gln Asn Ala Asn Leu Gln Thr Ala Ile Ala Gln
 435 440 445
 Ala Glu Gln His Gly Glu Met Ala Leu Lys Asp Ala Asn Ala Lys Leu
 450 455 460
 Gln Glu Leu Gln Ala Ala Leu Gln Gln Ala Lys Asp Asp Leu Ala Arg
 465 470 475 480
 Leu Leu Arg Asp Tyr Gln Glu Leu Met Asn Val Lys Leu Ala Leu Asp
 485 490 495
 Val Glu Ile Ala Thr Tyr Arg Lys Leu Leu Glu Gly Glu Glu Tyr Ser
 500 505 510
 Arg Met Ser Gly Glu Cys Pro Ser Ala Val Ser Ile Ser Val Val Ser
 515 520 525
 Ser Ser Thr Thr Ser Ala Ser Ala Gly Gly Tyr Gly Gly Gly Tyr Gly
 530 535 540
 Gly Gly Met Gly Gly Gly Leu Gly Gly Gly Phe Ser Ala Gly Gly Gly
 545 550 555 560
 Ser Gly Ile Gly Phe Gly Arg Gly Gly Gly Gly Ile Gly Gly Gly
 565 570 575
 Phe Gly Gly Gly Thr Ser Gly Phe Ser Gly Gly Ser Gly Phe Gly Ser
 580 585 590
 Ile Ser Gly Ala Arg Tyr Gly Val Ser Gly Gly Gly Phe Ser Ser Ala
 595 600 605
 Ser Asn Arg Gly Gly Ser Ile Lys Phe Ser Gln Ser Ser Gln Ser Ser
 610 615 620
 Gln Arg Tyr Ser Arg
 625
 <210> 465
 <211> 534
 <212> PRT
 <213> Homo sapiens

<400> 465

Met Ile Ala Arg Gln Gln Cys Val Arg Gly Gly Pro Arg Gly Phe Ser
 1 5 10 15
 Cys Gly Ser Ala Ile Val Gly Gly Gly Lys Arg Gly Ala Phe Ser Ser
 20 25 30
 Val Ser Met Ser Gly Gly Ala Gly Arg Cys Ser Ser Gly Gly Phe Gly
 35 40 45
 Ser Arg Ser Leu Tyr Asn Leu Arg Gly Asn Lys Ser Ile Ser Met Ser
 50 55 60
 Val Ala Gly Ser Arg Gln Gly Ala Cys Phe Gly Gly Ala Gly Gly Phe
 65 70 75 80
 Gly Thr Gly Gly Phe Gly Ala Gly Gly Phe Gly Ala Gly Phe Gly Thr
 85 90 95
 Gly Gly Phe Gly Gly Gly Phe Gly Gly Ser Phe Ser Gly Lys Gly Gly
 100 105 110
 Pro Gly Phe Pro Val Cys Pro Ala Gly Gly Ile Gln Glu Val Thr Ile
 115 120 125

Asn Gln Ser Leu Leu Thr Pro Leu His Val Glu Ile Asp Pro Glu Ile
 130 135 140
 Gln Lys Val Arg Thr Glu Arg Glu Gln Ile Lys Leu Leu Asn Asn
 145 150 155 160
 Lys Phe Ala Ser Phe Ile Asp Lys Val Gln Phe Leu Glu Gln Gln Asn
 165 170 175
 Lys Val Leu Glu Thr Lys Trp Asn Leu Leu Gln Gln Gln Thr Thr Thr
 180 185 190
 Thr Ser Ser Lys Asn Leu Glu Pro Leu Phe Glu Thr Tyr Leu Ser Val
 195 200 205
 Leu Arg Lys Gln Leu Asp Thr Leu Gly Asn Asp Lys Gly Arg Leu Gln
 210 215 220
 Ser Glu Leu Lys Thr Met Gln Asp Ser Val Glu Asp Phe Lys Thr Lys
 225 230 235 240
 Tyr Glu Glu Glu Ile Asn Lys Arg Thr Ala Ala Glu Asn Asp Phe Val
 245 250 255
 Val Leu Lys Lys Asp Val Asp Ala Ala Tyr Leu Asn Lys Val Glu Leu
 260 265 270
 Glu Ala Lys Val Asp Ser Leu Asn Asp Glu Ile Asn Phe Leu Lys Val
 275 280 285
 Leu Tyr Asp Ala Glu Leu Ser Gln Met Gln Thr His Val Ser Asp Thr
 290 295 300
 Ser Val Val Leu Ser Met Asp Asn Asn Arg Asn Leu Asp Leu Asp Ser
 305 310 315 320
 Ile Ile Ala Glu Val Arg Ala Gln Tyr Glu Glu Ile Ala Gln Arg Ser
 325 330 335
 Lys Ala Glu Ala Glu Ala Leu Tyr Gln Thr Lys Val Gln Gln Leu Gln
 340 345 350
 Ile Ser Val Asp Gln His Gly Asp Asn Leu Lys Asn Thr Lys Ser Glu
 355 360 365
 Ile Ala Glu Leu Asn Arg Met Ile Gln Arg Leu Arg Ala Glu Ile Glu
 370 375 380
 Asn Ile Lys Lys Gln Cys Gln Thr Leu Gln Val Ser Val Ala Asp Ala
 385 390 395 400
 Glu Gln Arg Gly Glu Asn Ala Leu Lys Asp Ala His Ser Lys Arg Val
 405 410 415
 Glu Leu Glu Ala Ala Leu Gln Gln Ala Lys Glu Glu Leu Ala Arg Met
 420 425 430
 Leu Arg Glu Tyr Gln Glu Leu Met Ser Val Lys Leu Ala Leu Asp Ile
 435 440 445
 Glu Ile Ala Thr Tyr Arg Lys Leu Leu Glu Gly Glu Glu Tyr Arg Met
 450 455 460
 Ser Gly Glu Cys Gln Ser Ala Val Ser Ile Ser Val Val Ser Gly Ser
 465 470 475 480
 Thr Ser Thr Gly Gly Ile Ser Gly Gly Leu Gly Ser Gly Ser Gly Phe
 485 490 495
 Gly Leu Ser Ser Gly Phe Gly Ser Gly Ser Gly Ser Gly Phe Gly Phe
 500 505 510
 Gly Gly Ser Val Ser Gly Ser Ser Ser Lys Ile Ile Ser Thr Thr
 515 520 525
 Thr Leu Asn Lys Arg Arg
 530

<210> 466

<211> 483

<212> PRT

<213> Homo sapiens

<400> 466

Met Ser Ile Arg Val Thr Gln Lys Ser Tyr Lys Val Ser Thr Ser Gly
 1 5 10 15

Pro Arg Ala Phe Ser Ser Arg Ser Tyr Thr Ser Gly Pro Gly Ser Arg
 Ile Ser Ser Ser Ser Phe Ser Arg Val Gly Ser Ser Asn Phe Arg Gly
 Gly Leu Gly Gly Gly Tyr Gly Gly Ala Ser Gly Met Gly Gly Ile Thr
 Ala Val Thr Val Asn Gln Ser Leu Leu Ser Pro Leu Val Leu Glu Val
 Asp Pro Asn Ile Gln Ala Val Arg Thr Gln Glu Lys Glu Gln Ile Lys
 Thr Leu Asn Asn Lys Phe Ala Ser Phe Ile Asp Lys Val Arg Phe Leu
 Glu Gln Gln Asn Lys Met Leu Glu Thr Lys Trp Ser Leu Leu Gln Gln
 Gln Lys Thr Ala Arg Ser Asn Met Asp Asn Met Phe Glu Ser Tyr Ile
 Asn Asn Leu Arg Arg Gln Leu Glu Thr Leu Gly Gln Glu Lys Leu Lys
 Leu Glu Ala Glu Leu Gly Asn Met Gln Gly Leu Val Glu Asp Phe Lys
 Asn Lys Tyr Glu Asp Glu Ile Asn Lys Arg Thr Glu Met Glu Asn Glu
 Phe Val Leu Ile Lys Lys Asp Val Asp Glu Ala Tyr Met Asn Lys Val
 Glu Leu Glu Ser Arg Leu Glu Gly Leu Thr Asp Glu Ile Asn Phe Leu
 Arg Gln Leu Tyr Glu Glu Ile Arg Glu Leu Gln Ser Gln Ile Ser
 Asp Thr Ser Val Val Leu Ser Met Asp Asn Ser Arg Ser Leu Asp Met
 Asp Ser Ile Ile Ala Glu Val Lys Ala Gln Tyr Glu Asp Ile Ala Asn
 Arg Ser Arg Ala Glu Ala Glu Ser Met Tyr Gln Ile Lys Tyr Glu Glu
 Leu Gln Ser Leu Ala Gly Lys His Gly Asp Asp Leu Arg Arg Thr Lys
 Thr Glu Ile Ser Glu Met Asn Arg Asn Ile Ser Arg Leu Gln Ala Glu
 Ile Glu Gly Leu Lys Gly Gln Arg Ala Ser Leu Glu Ala Ala Ile Ala
 Asp Ala Glu Gln Arg Gly Glu Leu Ala Ile Lys Asp Ala Asn Ala Lys
 Leu Ser Glu Leu Glu Ala Ala Leu Gln Arg Ala Lys Gln Asp Met Ala
 Arg Gln Leu Arg Glu Tyr Gln Glu Leu Met Asn Val Lys Leu Ala Leu
 Asp Ile Glu Ile Ala Thr Tyr Arg Lys Leu Leu Glu Gly Glu Glu Ser
 Arg Leu Glu Ser Gly Met Gln Asn Met Ser Ile His Thr Lys Thr Thr
 Ser Gly Tyr Ala Gly Gly Leu Ser Ser Ala Tyr Gly Gly Leu Thr Ser
 Pro Gly Leu Ser Tyr Ser Leu Gly Ser Ser Phe Gly Ser Gly Ala Gly
 Ser Ser Ser Phe Ser Arg Thr Ser Ser Ser Arg Ala Val Val Lys
 Lys Ile Glu Thr Arg Asp Gly Lys Leu Val Ser Glu Ser Ser Asp Val
 Leu Pro Lys

<210> 467

<211> 430

<212> PRT

<213> Homo sapiens

<400> 467

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Met Ser Phe Thr Thr Arg Ser Thr Phe Ser Thr Asn Tyr Arg Ser Leu
1      5      10      15
Gly Ser Val Gln Ala Pro Ser Tyr Gly Ala Arg Pro Val Ser Ser Ala
20      25      30
Ala Ser Val Tyr Ala Gly Ala Gly Ser Gly Ser Arg Ile Ser Val
35      40      45
Ser Arg Ser Thr Ser Phe Arg Gly Gly Met Gly Ser Gly Gly Leu Ala
50      55      60
Thr Gly Ile Ala Gly Gly Leu Ala Gly Met Gly Gly Ile Gln Asn Glu
65      70      75      80
Lys Glu Thr Met Gln Ser Leu Asn Asp Arg Leu Ala Ser Tyr Leu Asp
85      90      95
Arg Val Arg Ser Leu Glu Thr Glu Asn Arg Arg Leu Glu Ser Lys Ile
100     105     110
Arg Glu His Leu Glu Lys Lys Gly Pro Gln Val Arg Asp Trp Ser His
115     120     125
Tyr Phe Lys Ile Ile Glu Asp Leu Arg Ala Gln Ile Phe Ala Asn Thr
130     135     140
Val Asp Asn Ala Arg Ile Val Leu Gln Ile Asp Asn Ala Arg Leu Ala
145     150     155     160
Ala Asp Asp Phe Arg Val Lys Tyr Glu Thr Glu Leu Ala Met Arg Gln
165     170     175
Ser Val Glu Asn Asp Ile His Gly Leu Arg Lys Val Ile Asp Asp Thr
180     185     190
Asn Ile Thr Arg Leu Gln Leu Glu Thr Glu Ile Glu Ala Leu Lys Glu
195     200     205
Glu Leu Leu Phe Met Lys Lys Asn His Glu Glu Glu Val Lys Gly Leu
210     215     220
Gln Ala Gln Ile Ala Ser Ser Gly Leu Thr Val Glu Val Asp Ala Pro
225     230     235     240
Lys Ser Gln Asp Leu Ala Lys Ile Met Ala Asp Ile Arg Ala Gln Tyr
245     250     255
Asp Glu Leu Ala Arg Lys Asn Arg Glu Glu Leu Asp Lys Tyr Trp Ser
260     265     270
Gln Gln Ile Glu Glu Ser Thr Thr Val Val Thr Thr Gln Ser Ala Glu
275     280     285
Val Gly Ala Ala Glu Thr Thr Leu Thr Glu Leu Arg Arg Thr Val Gln
290     295     300
Ser Leu Glu Ile Asp Leu Asp Ser Met Arg Asn Leu Lys Ala Ser Leu
305     310     315     320
Glu Asn Ser Leu Arg Glu Val Glu Ala Arg Tyr Ala Leu Gln Met Glu
325     330     335
Gln Leu Asn Gly Ile Leu Leu His Leu Glu Ser Glu Leu Ala Gln Thr
340     345     350
Arg Ala Glu Gly Gln Arg Gln Ala Gln Glu Tyr Glu Ala Leu Leu Asn
355     360     365
Ile Lys Val Lys Leu Glu Ala Glu Ile Ala Thr Tyr Arg Arg Leu Leu
370     375     380
Glu Asp Gly Glu Asp Phe Asn Leu Gly Asp Ala Leu Asp Ser Ser Asn
385     390     395     400
Ser Met Gln Thr Ile Gln Lys Thr Thr Thr Arg Arg Ile Val Asp Gly
405     410     415
Lys Val Val Ser Glu Thr Asn Asp Thr Lys Val Leu Arg His
420     425     430

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<210> 468

<211> 392

<212> PRT

<213> Homo sapiens

<400> 468
 Met Val Ala Arg Val Gly Leu Leu Leu Arg Ala Leu Gln Leu Leu Leu
 1 5 10 15
 Trp Gly His Leu Asp Ala Gln Pro Ala Glu Arg Gly Gly Gln Glu Leu
 20 25 30
 Arg Lys Glu Ala Glu Ala Phe Leu Glu Lys Tyr Gly Tyr Leu Asn Glu
 35 40 45
 Gln Val Pro Lys Ala Pro Thr Ser Thr Arg Phe Ser Asp Ala Ile Arg
 50 55 60
 Ala Phe Gln Trp Val Ser Gln Leu Pro Val Ser Gly Val Leu Asp Arg
 65 70 75 80
 Ala Thr Leu Arg Gln Met Thr Arg Pro Arg Cys Gly Val Thr Asp Thr
 85 90 95
 Asn Ser Tyr Ala Ala Trp Ala Glu Arg Ile Ser Asp Leu Phe Ala Arg
 100 105 110
 His Arg Thr Lys Met Arg Arg Lys Lys Arg Phe Ala Lys Gln Gly Asn
 115 120 125
 Lys Trp Tyr Lys Gln His Leu Ser Tyr Arg Leu Val Asn Trp Pro Glu
 130 135 140
 His Leu Pro Glu Pro Ala Val Arg Gly Ala Val Arg Ala Ala Phe Gln
 145 150 155 160
 Leu Trp Ser Asn Val Ser Ala Leu Glu Phe Trp Glu Ala Pro Ala Thr
 165 170 175
 Gly Pro Ala Asp Ile Arg Leu Thr Phe Phe Gln Gly Asp His Asn Asp
 180 185 190
 Gly Leu Gly Asn Ala Phe Asp Gly Pro Gly Gly Ala Leu Ala His Ala
 195 200 205
 Phe Leu Pro Arg Arg Gly Glu Ala His Phe Asp Gln Asp Glu Arg Trp
 210 215 220
 Ser Leu Ser Arg Arg Arg Gly Arg Asn Leu Phe Val Val Leu Ala His
 225 230 235 240
 Glu Ile Gly His Thr Leu Gly Leu Thr His Ser Pro Ala Pro Arg Ala
 245 250 255
 Leu Met Ala Pro Tyr Tyr Lys Arg Leu Gly Arg Asp Ala Leu Leu Ser
 260 265 270
 Trp Asp Asp Val Leu Ala Val Gln Ser Leu Tyr Gly Lys Pro Leu Gly
 275 280 285
 Gly Ser Val Ala Val Gln Leu Pro Gly Lys Leu Phe Thr Asp Phe Glu
 290 295 300
 Thr Trp Asp Ser Tyr Ser Pro Gln Gly Arg Arg Pro Glu Thr Gln Gly
 305 310 315 320
 Pro Lys Tyr Cys His Ser Ser Phe Asp Ala Ile Thr Val Asp Arg Gln
 325 330 335
 Gln Gln Leu Tyr Ile Phe Lys Gly Ser His Phe Trp Glu Val Ala Ala
 340 345 350
 Asp Gly Asn Val Ser Glu Pro Arg Pro Leu Gln Glu Arg Trp Val Gly
 355 360 365
 Leu Pro Pro Asn Ile Glu Ala Ala Ala Val Ser Leu Asn Asp Gly Asp
 370 375 380
 Phe Tyr Phe Phe Lys Val Gln Ser
 385 390

<210> 469

<211> 851

<212> PRT

<213> Homo sapiens

<400> 469

Met	Ala	Gln	Trp	Glu	Met	Leu	Gln	Asn	Leu	Asp	Ser	Pro	Phe	Gln	Asp
1				5					10					15	
Gln	Leu	His	Gln	Leu	Tyr	Ser	His	Ser	Leu	Leu	Pro	Val	Asp	Ile	Arg
			20					25					30		
Gln	Tyr	Leu	Ala	Val	Trp	Ile	Glu	Asp	Gln	Asn	Trp	Gln	Glu	Ala	Ala
		35					40					45			
Leu	Gly	Ser	Asp	Asp	Ser	Lys	Ala	Thr	Met	Leu	Phe	Phe	His	Phe	Leu
	50					55					60				
Asp	Gln	Leu	Asn	Tyr	Glu	Cys	Gly	Arg	Cys	Ser	Gln	Asp	Pro	Glu	Ser
65					70					75					80
Leu	Leu	Leu	Gln	His	Asn	Leu	Arg	Lys	Phe	Cys	Arg	Asp	Ile	Gln	Pro
				85					90					95	
Phe	Ser	Gln	Asp	Pro	Thr	Gln	Leu	Ala	Glu	Met	Ile	Phe	Asn	Leu	Leu
			100					105					110		
Leu	Glu	Glu	Lys	Arg	Ile	Leu	Ile	Gln	Ala	Gln	Arg	Ala	Gln	Leu	Glu
		115					120					125			
Gln	Gly	Glu	Pro	Val	Leu	Glu	Thr	Pro	Val	Glu	Ser	Gln	Gln	His	Glu
	130					135					140				
Ile	Glu	Ser	Arg	Ile	Leu	Asp	Leu	Arg	Ala	Met	Met	Glu	Lys	Leu	Val
145					150					155					160
Lys	Ser	Ile	Ser	Gln	Leu	Lys	Asp	Gln	Gln	Asp	Val	Phe	Cys	Phe	Arg
				165				170						175	
Tyr	Lys	Ile	Gln	Ala	Lys	Gly	Lys	Thr	Pro	Ser	Leu	Asp	Pro	His	Gln
			180					185					190		
Thr	Lys	Glu	Gln	Lys	Ile	Leu	Gln	Glu	Thr	Leu	Asn	Glu	Leu	Asp	Lys
		195					200					205			
Arg	Arg	Lys	Glu	Val	Leu	Asp	Ala	Ser	Lys	Ala	Leu	Leu	Gly	Arg	Leu
	210					215					220				
Thr	Thr	Leu	Ile	Glu	Leu	Leu	Leu	Pro	Lys	Leu	Glu	Glu	Trp	Lys	Ala
225					230					235					240
Gln	Gln	Gln	Lys	Ala	Cys	Ile	Arg	Ala	Pro	Ile	Asp	His	Gly	Leu	Glu
				245					250					255	
Gln	Leu	Glu	Thr	Trp	Phe	Thr	Ala	Gly	Ala	Lys	Leu	Leu	Phe	His	Leu
			260					265					270		
Arg	Gln	Leu	Leu	Lys	Glu	Leu	Lys	Gly	Leu	Ser	Cys	Leu	Val	Ser	Tyr
		275					280					285			
Gln	Asp	Asp	Pro	Leu	Thr	Lys	Gly	Val	Asp	Leu	Arg	Asn	Ala	Gln	Val
	290					295					300				
Thr	Glu	Leu	Leu	Gln	Arg	Leu	Leu	His	Arg	Ala	Phe	Val	Val	Glu	Thr
305					310					315					320
Gln	Pro	Cys	Met	Pro	Gln	Thr	Pro	His	Arg	Pro	Leu	Ile	Leu	Lys	Thr
				325					330					335	
Gly	Ser	Lys	Phe	Thr	Val	Arg	Thr	Arg	Leu	Leu	Val	Arg	Leu	Gln	Glu
			340					345					350		
Gly	Asn	Glu	Ser	Leu	Thr	Val	Glu	Val	Ser	Ile	Asp	Arg	Asn	Pro	Pro
	355						360					365			
Gln	Leu	Gln	Gly	Phe	Arg	Lys	Phe	Asn	Ile	Leu	Thr	Ser	Asn	Gln	Lys
	370					375					380				
Thr	Leu	Thr	Pro	Glu	Lys	Gly	Gln	Ser	Gln	Gly	Leu	Ile	Trp	Asp	Phe
385					390					395					400
Gly	Tyr	Leu	Thr	Leu	Val	Glu	Gln	Arg	Ser	Gly	Gly	Ser	Gly	Lys	Gly
			405						410					415	
Ser	Asn	Lys	Gly	Pro	Leu	Gly	Val	Thr	Glu	Glu	Leu	His	Ile	Ile	Ser
			420					425					430		
Phe	Thr	Val	Lys	Tyr	Thr	Tyr	Gln	Gly	Leu	Lys	Gln	Glu	Leu	Lys	Thr
		435					440					445			
Asp	Thr	Leu	Pro	Val	Val	Ile	Ile	Ser	Asn	Met	Asn	Gln	Leu	Ser	Ile
	450					455					460				
Ala	Trp	Ala	Ser	Val	Leu	Trp	Phe	Asn	Leu	Leu	Ser	Pro	Asn	Leu	Gln
465					470					475					480
Asn	Gln	Gln	Phe	Phe	Ser	Asn	Pro	Pro	Lys	Ala	Pro	Trp	Ser	Leu	Leu
			485						490					495	
Gly	Pro	Ala	Leu	Ser	Trp	Gln	Phe	Ser	Ser	Tyr	Val	Gly	Arg	Gly	Leu
			500					505					510		

Asn Ser Asp Gln Leu Ser Met Leu Arg Asn Lys Leu Phe Gly Gln Asn
 515 520 525
 Cys Arg Thr Glu Asp Pro Leu Leu Ser Trp Ala Asp Phe Thr Lys Arg
 530 535 540
 Glu Ser Pro Pro Gly Lys Leu Pro Phe Trp Thr Trp Leu Asp Lys Ile
 545 550 555 560
 Leu Glu Leu Val His Asp His Leu Lys Asp Leu Trp Asn Asp Gly Arg
 565 570 575
 Ile Met Gly Phe Val Ser Arg Ser Gln Glu Arg Arg Leu Leu Lys Lys
 580 585 590
 Thr Met Ser Gly Thr Phe Leu Leu Arg Phe Ser Glu Ser Ser Glu Gly
 595 600 605
 Gly Ile Thr Cys Ser Trp Val Glu His Gln Asp Asp Asp Lys Val Leu
 610 615 620
 Ile Tyr Ser Val Gln Pro Tyr Thr Lys Glu Val Leu Gln Ser Leu Pro
 625 630 635 640
 Leu Thr Glu Ile Ile Arg His Tyr Gln Leu Leu Thr Glu Glu Asn Ile
 645 650 655
 Pro Glu Asn Pro Leu Arg Phe Leu Tyr Pro Arg Ile Pro Arg Asp Glu
 660 665 670
 Ala Phe Gly Cys Tyr Tyr Gln Glu Lys Val Asn Leu Gln Glu Arg Arg
 675 680 685
 Lys Tyr Leu Lys His Arg Leu Ile Val Val Ser Asn Arg Gln Val Asp
 690 695 700
 Glu Leu Gln Gln Pro Leu Glu Leu Lys Pro Glu Pro Glu Leu Glu Ser
 705 710 715 720
 Leu Glu Leu Glu Leu Gly Leu Val Pro Glu Pro Glu Leu Ser Leu Asp
 725 730 735
 Leu Glu Pro Leu Leu Lys Ala Gly Leu Asp Leu Gly Pro Glu Leu Glu
 740 745 750
 Ser Val Leu Glu Ser Thr Leu Glu Pro Val Ile Glu Pro Thr Leu Cys
 755 760 765
 Met Val Ser Gln Thr Val Pro Glu Pro Asp Gln Gly Pro Val Ser Gln
 770 775 780
 Pro Val Pro Glu Pro Asp Leu Pro Cys Asp Leu Arg His Leu Asn Thr
 785 790 795 800
 Glu Pro Met Glu Ile Phe Arg Asn Cys Val Lys Ile Glu Glu Ile Met
 805 810 815
 Pro Asn Gly Asp Pro Leu Leu Ala Gly Gln Asn Thr Val Asp Glu Val
 820 825 830
 Tyr Val Ser Arg Pro Ser His Phe Tyr Thr Asp Gly Pro Leu Met Pro
 835 840 845
 Ser Asp Phe
 850

<210> 470

<211> 335

<212> PRT

<213> Homo sapiens

<400> 470

Met Gly Lys Val Lys Val Gly Val Asn Gly Phe Gly Arg Ile Gly Arg
 1 5 10 15
 Leu Val Thr Arg Ala Ala Phe Asn Ser Gly Lys Val Asp Ile Val Ala
 20 25 30
 Ile Asn Asp Pro Phe Ile Asp Leu Asn Tyr Met Val Tyr Met Phe Gln
 35 40 45
 Tyr Asp Ser Thr His Gly Lys Phe His Gly Thr Val Lys Ala Glu Asn
 50 55 60
 Gly Lys Leu Val Ile Asn Gly Asn Pro Ile Thr Ile Phe Gln Glu Arg
 65 70 75 80

- 279 -

Asp	Pro	Ser	Lys	Ile	Lys	Trp	Gly	Asp	Ala	Gly	Ala	Glu	Tyr	Val	Val	
				85					90					95		
Glu	Ser	Thr	Gly	Val	Phe	Thr	Thr	Met	Glu	Lys	Ala	Gly	Ala	His	Leu	
			100					105						110		
Gln	Gly	Gly	Ala	Lys	Arg	Val	Ile	Ile	Ser	Ala	Pro	Ser	Ala	Asp	Ala	
		115					120					125				
Pro	Met	Phe	Val	Met	Gly	Val	Asn	His	Glu	Lys	Tyr	Asp	Asn	Ser	Leu	
	130					135					140					
Lys	Ile	Ile	Ser	Asn	Ala	Ser	Cys	Thr	Thr	Asn	Cys	Leu	Ala	Pro	Leu	
145					150					155					160	
Ala	Lys	Val	Ile	His	Asp	Asn	Phe	Gly	Ile	Val	Glu	Gly	Leu	Met	Thr	
				165					170						175	
Thr	Val	His	Ala	Ile	Thr	Ala	Thr	Gln	Lys	Thr	Val	Asp	Gly	Pro	Ser	
			180					185					190			
Gly	Lys	Leu	Trp	Arg	Asp	Gly	Arg	Gly	Ala	Leu	Gln	Asn	Ile	Ile	Pro	
		195					200					205				
Ala	Ser	Thr	Gly	Ala	Ala	Lys	Ala	Val	Gly	Lys	Val	Ile	Pro	Glu	Leu	
	210					215					220					
Asn	Gly	Lys	Leu	Thr	Gly	Met	Ala	Phe	Arg	Val	Pro	Thr	Ala	Asn	Val	
225					230					235					240	
Ser	Val	Val	Asp	Leu	Thr	Cys	Arg	Leu	Glu	Lys	Pro	Ala	Lys	Tyr	Asp	
			245						250					255		
Asp	Ile	Lys	Lys	Val	Val	Lys	Gln	Ala	Ser	Glu	Gly	Pro	Leu	Lys	Gly	
			260					265					270			
Ile	Leu	Gly	Tyr	Thr	Glu	His	Gln	Val	Val	Ser	Ser	Asp	Phe	Asn	Ser	
		275					280					285				
Asp	Thr	His	Ser	Ser	Thr	Phe	Asp	Ala	Gly	Ala	Gly	Ile	Ala	Leu	Asn	
	290					295					300					
Asp	His	Phe	Val	Lys	Leu	Ile	Ser	Trp	Tyr	Asp	Asn	Glu	Phe	Gly	Tyr	
305					310					315					320	
Ser	Asn	Arg	Val	Val	Asp	Leu	Met	Ala	His	Met	Ala	Ser	Lys	Glu		
				325					330					335		

<210> 471

<211> 22

<212> DNA

<213> Artificial Sequence

<220>

<223> NAP4 Probe

<400> 471

tccgcctcag tcgcctcttt cg

<210> 472

<211> 19

<212> DNA

<213> Artificial Sequence

<220>

<223> NAP4 FOR PRIMER

<400> 472

tcggaagggc tccttcaaa

<210> 473

<211> 19

<212> DNA

<213> Artificial Sequence

<220>

<223> NAP4 REV PRIMER

<400> 473

caccggttgca gctcttggt

<210> 474

19

<211> 34

<212> DNA

<213> Artificial Sequence

<220>

<223> MRLP45 Probe

<400> 474

ctcccatgcc cctcatgcta taaaaagaac tacc

<210> 475

34

<211> 20

<212> DNA

<213> Artificial Sequence

<220>

<223> MRLP45 FOR PRIMER

<400> 475

ggctgctgga agctttgaag

<210> 476

20

<211> 21

<212> DNA

<213> Artificial Sequence

<220>

<223> MRLP45 REV PRIMER

<400> 476

tgagcaggat gggagagaac a

<210> 477

21

<211> 22

<212> DNA

<213> Artificial Sequence

<220>

<223> TCF2 Probe

<400> 477

caaaagctgg ccatggacgc ct

22

<210> 478

<211> 20

<212> DNA

<213> Artificial Sequence

<220>

<223> TCF2 FOR PRIMER

<400> 478

gcaggaagga ggaggcattc

20

<210> 479

<211> 21

<212> DNA

<213> Artificial Sequence

<220>

<223> TCF2 REV PRIMER

<400> 479

caggctgtga gtctggttgg a

21

<210> 480

<211> 25

<212> DNA

<213> Artificial Sequence

<220>

<223> ROK1 Probe

<400> 480

cagctggcctt ccattttcct ggcct

25

<210> 481

<211> 21

<212> DNA

<213> Artificial Sequence

<220>

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